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The Medical Clinics of Chicago

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PUBLISHERS' ANNOUNCEMENT

In presenting to the profession the MEDICAL CLINICS OF CHICAGO we are responding to a demand by the physicians in all parts of the country for a work of this character coming at regular intervals.

It will be recognized that we have secured the clinics of some of the best clinical teachers in Chicago in every department of INTERNAL MEDICINE, including Neurology, Pediatrics, *x*-Ray Therapy, etc. We hope in this way to give the profession in each number a series of cases representing all branches of internal medicine which shall be word photographs of the actual up-to-date management of each case in its important phases by the attending physician.

These records of diseases will be gathered at the bedside of patients and in the amphitheaters of the leading hospitals of Chicago, and will include, of course, the histories, examination, description of diagnostic methods, treatment, and such other data as will be desirable to afford the reader a complete and satisfactory understanding of the subject.

This first number speaks for itself, and we hope will prove a correct interpretation of our understanding of the needs and desires of the doctors of this country—both the general practitioners and the specialists—for a series of actual clinical teaching in the field of internal medicine.

The second number, among other things, will contain clinics on Tuberculous Meningitis in Children (with full description of

the Permanganate of Potash Test), by Dr. Isaac A. Abt; Heart Disease in Pregnancy and the Indications for Therapeutic Abortion, with a full discussion of the question of marriage in relation to each type of cardiopathy in both the male and female; also a clinic on Infantilism, with complete classification and discussion of the rôle of the internal secretions, by Dr. Frederick Tice; the first of a series of clinics on the subject of Neuritis in its various phases, by Dr. Ralph C. Hamill; the first of a series of clinics dealing in particular with The Diagnostic and Prognostic Significance of Eye Lesions in Relation to General Systemic Disorders, by Dr. Richard J. Tivnen; Duodenal Ulcer and Gastric Carcinoma, by Dr. Charles L. Mix; *x*-Rays in Epithelioma, by Dr. William Allen Pusey, etc., etc.

The
Medical Clinics of Chicago

VOLUME 1 NUMBER 1

CLINIC OF DR. CHARLES L. MIX

MERCY HOSPITAL

LUNG ABSCESS WITH THE PICTURE OF TUBERCULOSIS

THE patient is a Sister, a nun, thirty-one years of age; her father died at the age of seventy-five of cancer of the stomach; her mother at forty-five years of age of consumption (?). She has seven sisters and three brothers who are living and well. There is some doubt of what the mother actually died of; the belief of some of the daughters, who are nurses, is that the mother did not die of tuberculosis. It has to be left an open question.

The patient was perfectly well until she was twelve years of age, when she had scarlet fever. It was a severe case of scarlet fever and was associated with otitis media; an abscess formed in the cavity of the middle ear; the ear-drum ruptured and the ear discharged for a while. She was kept in bed six weeks, when, she said, she suffered a relapse. This was six weeks from the time of the original rash. She, however, made a perfect recovery after a long period of illness. There were no renal complications with the scarlet fever. Her ear also got entirely well, so that she hears perfectly.

After that she remained well until the year 1910. In the fall of that year, that is to say, about the first of November, 1910, she was exposed to a peculiar epidemic of mumps and diphtheria. She says she had the mumps and thereafter developed a case of diphtheria. She had the mumps about the first of November, 1910, and three weeks thereafter, about the 21st

of November, was taken down with diphtheria. It does not seem to have been of a very severe type, yet it produced a marked effect upon the myocardium. Indeed, the only effect which diphtheria can produce upon the heart is a toxic myocarditis. Diphtheria is a toxemia, not a bacteriemia. Hence, it can give rise only to myocardial poisoning, but not to endocarditis. In these cardiac cases there is usually a slowing of the pulse and arhythmia. Those patients who die of cardiac paralysis following diphtheria are very apt to show a marked slowing of the pulse before death. The toxemia which produces a slowing of the pulse also produces a loss of the knee-jerks, and in those cases in which the heart is severely affected you almost invariably find that the knee-jerks are lost first. Indeed, one can know in a diphtheria case whether the heart is likely to be adversely affected, by the behavior of the reflexes, so that any one caring for a case of diphtheria should always pay close attention to the presence or absence of the knee-jerks.

Another thing about this disease which is very characteristic is that it always takes time for the toxemia to produce its effect, and, consequently, you do not perceive its influence on the heart until the patient is approaching a healthy state as regards the throat and the diphtheria. The doctor oftentimes thinks that the child is all right, and he permits it to get up and play, and it falls dead. In other words, these cardiac disturbances which take place as a sequence of diphtheria ordinarily occur in the period of convalescence after sufficient time has elapsed for the toxemia to have produced a degenerative effect upon the myocardium.

So in this case, after her diphtheria was over, she noticed the arhythmia. She states that her heart had a one-, two-, three-beat and then a stop, and then one, two, three, and then a stop; in other words, she had an ordinary cardiac arhythmia, and of the type suggesting a mild degree of heart-block. This took place evidently some time in December. She speaks of her second cardiac irregularity as occurring about Christmas of that same year, 1910.

Now (addressing Patient), after that you were well for a period of about three years' time?

PATIENT: Yes.

DR. MIX (continuing): And then Christmas, 1913, she began to run down. It was noticed by her friends that she was not in as good condition as she had been up to that time. Her condition remained under par until the following July.

In June, 1914, she caught cold in her chest. She had a cold on her lungs, as she expressed it, but it lasted only a week; it got well and then she remained in her usual state until it was decided that her tonsils should be removed. The tonsils were examined, and it was found that following the diphtheria they had never been restored to their original healthy state. Her run-down condition was attributed to the chronic infection of these tonsils, and the tonsils were, therefore, removed on the 15th day of July.

Then something new developed: Her present condition came about. You can see, I think, as we review the history a possible connection between the removal of the tonsils and her subsequent illness. A week after the tonsils were removed the patient again took a cold. She said that "the cold settled on her lungs."

The attending physician, on account of the antecedent history and her run-down condition of many months preceding the removal of the tonsils, was of the opinion that the trouble was probably tuberculosis. As the patient says, "he was very suspicious that the trouble was of a tuberculous nature." He examined the sputum, and reported that tubercle bacilli were present. She was told that the trouble with her chest was tuberculous, and that there was no possibility whatever of infection of the lung from the tonsils; in other words, that there was no connection between the removal of the tonsils and the infection in the lung.

Now, during August this cold on her chest rapidly became very much worse. On August 15th her temperature went as high as 102° F., and she began to cough and, furthermore, began to expectorate a yellowish sputum, the odor of which was very

offensive. It was so offensive, she says, that it was almost impossible for other people to remain in the same room with her. *The high degree of offensiveness of odor should make one certain that it could not possibly have been an ordinary case of tuberculosis.*

The temperature fell during the month of August, and the patient came to this (Mercy) hospital the first part of September. She remained in this hospital in another service for three days for observation and diagnosis. The diagnosis of tuberculosis was confirmed by a colleague on account of the history of tubercle bacilli having been found, although in this laboratory (Mercy Hospital) the sputum was examined and the tubercle bacilli were *not* found. At this time she was running no temperature and her pulse at the time of entrance was 90, but with the rest in bed it quickly fell to 70 and 74, and she felt fairly well. She then returned to her home, where she remained until she re-entered the hospital.

After she had been at home ten days she took a hot bath and when she had finished she felt very weak, which is, of course, to be expected. Since she evidently had a severe infection and since the heart was to some degree affected, it is perfectly evident that a hot bath was the wrong thing to take. After taking the hot bath she went to bed, but soon woke up and coughed, and at every cough she spat up bright red blood. She does not know the exact quantity because, she said, it was not collected. The cough subsided during the night and she went to sleep.

In the morning she expectorated yellow mucus streaked with blood. The yellow mucus also was very foul smelling. She did not feel any weaker than she ordinarily felt, but rested that day in a chair. Upon retiring that night the pulse became very weak, and the attending nurse was unable to count the beats. The calves of her leg were cold to the touch, indicating a marked degree of shock, possibly due to loss of blood. The next day she remained in bed. The next evening, that is, the evening of the second day, her heart-beat was very rapid; at that time both strychnin and digitalis were given to her, and her physician stated to her that she had a "galloping heart."

Now comes another episode in her history, a very important one: On a Sunday she was seized with a severe pain under the right scapula and her temperature went rapidly to 102° F. at 11 A. M., and stayed up until 2 P. M.; it then began to fall again. The physician who was called ordered an ice-bag to be placed in front of her heart, and 15 drops of tincture of digitalis to be given to her four times a day. She had taken four doses when, the next morning, she noticed a jerking of the heart, and the physician reduced the digitalis to 10 drops four times a day. She remained then in a condition of serious illness, with coughing and expectoration of the foul-smelling sputum, until two weeks before entering the hospital.

Two weeks before entering the hospital she got out of bed and stayed up four days—Saturday, Sunday, Monday, and Tuesday. Tuesday night she became rather excited and her pulse-rate became very rapid, probably in part due to the perception on her part of the heart action. That night she "felt a rush of blood to the left side of the heart," and then she had a coughing spell which filled two handkerchiefs with pus and mucus. She was given $\frac{1}{4}$ grain of codein. She did not expectorate that night, but she coughed continuously from that time to the time she entered the hospital, when she was spitting up mucus streaked with blood and pus. She remained in bed until she entered the hospital, which was on the 11th of November, 1914. From that time we have a little more accurate information in regard to her case.

A blood examination which was made on the 12th day of November, 1914, showed the hemoglobin to be 75 per cent.; the red count, 4,480,000, and the white count, 12,800—a comparatively good finding. The leukocytosis raises a presumption against uncomplicated tuberculosis.

The sputum which was examined on the 12th day of November, the day after entrance, was found to contain no tubercle bacilli, and the urinalysis showed no sugar, no red blood-cells, and two to four white cells to a field, using the $\frac{1}{16}$-inch objective. The total quantity was 480 c.c., and the specific gravity 1020.

An examination of the urine made on the 13th of November showed the same findings, and the specific gravity was also 1020. On the 13th the sputum was again examined and showed no tubercle bacilli. No tubercle bacilli were found on the 14th and on the 16th of November. A blood-culture was made on the 14th of November and was found to be negative. The sputum was examined again on the 21st and no tubercle bacilli found. On the 24th we ordered the sputum to be examined by the antiformin method, which is a very exact method of finding tubercle bacilli, but none were found. This was repeated on the 25th and none found; no tubercle bacilli were found on the 27th of November, nor on the 2d of December, nor at subsequent times when the sputum was examined.

There is one rather interesting finding which I want to call to your attention, that is, the enlargement of the terminal phalanges of the fingers. You will notice that this second finger, especially its terminal phalanx, is very much larger than the middle finger; and you will notice that the nail itself is curved. (Indicating on patient.) The nail is much wider than it ought to be and it is much smoother, and is curved like the beak of a parrot. The first, second and third fingers, and the thumb show more changes than does the little finger. The other hand (indicating) shows about the same sort of thing. You will notice the second and third fingers on this hand (right) show the maximum amount of enlargement. That condition is rather pathognomonic of a certain state of affairs. It occurs practically only in those cases where there is an abscess of the lung, or where there is a congenital heart lesion or one acquired early in life. Contrary to general opinion, this change in the ends of the fingers and in the nails is a thing which can be acquired rapidly.

Dr. Mix (addressing Patient): Your fingers in September were just as they always have been?

Patient: In October also. The enlargement began between October and November.

Dr. Mix: So that now, in March, they have reached these proportions. You see that the process is not necessarily, as sometimes intimated, the result of a congenital heart lesion or

Figs. 1 and 2.—Photographs of hands showing rapid development of clubbing in five months' time. Note the third fingers especially, and the shiny and curved "parrot-beaked" finger-nails, illustrating Marie's *osteoarthropathie pulmonique*.

pulmonary abscess occurring only in infancy or early childhood. It can come on at any time in life.

I once had a man under my care with emphysema, bronchitis, and asthma—a combination which is not at all infrequent. The chronic bronchitis was of two years' duration. There was a good deal of expectoration. He had some enlarged finger-nails. As I looked at him I happened casually to pick up his hand. I said, "Those finger-nails are a part of the disease." He replied: "I always thought so, but the doctors thought not. The reason I thought they were is that I never had them till I got this lung trouble." These changes can take place, then, in the ends of the fingers of individuals even past fifty years of age with comparative rapidity. In this particular case it began to take place in three or four months from the time the trouble appeared in the chest.

Now, when we came to examine this patient (referring to patient present) we were struck first with the fact that *she did not have an involvement of the apices of her lungs.* There was no dulness over either of the apices. There were no râles in either of the apices. There were no prolongations of expiratory murmurs in either apex.

Now, *the ordinary signs of pulmonary tuberculosis* are just those three things I have spoken of:

Dulness in an apex;

Prolongation of the expiratory murmur, which is another way of saying that there is bronchial breathing of a definite sort; and

The presence of one or more clicking râles.

The patient did, however, show dulness upon percussing her lungs in the back. There was an area which was found extremely dull situated a little below the angle of the scapula on the right side. (Demonstrated on patient.)

You will remember that on a certain Sunday she was suddenly seized with very severe pain under the angle of the scapula. From that time her condition became very much worse than it had been previous to that time. It was upon that Sunday that this area, undoubtedly by a metastatic infection, becamed involved. This area of dulness which we have found is associated

with a very intense bronchial breathing and with râles which are of a coarse variety. They are not the bubbling râles, such as might be found in a cavity, but coarse râles.

There is also in her case another very important finding, and that is a "cracked-pot" sound; that is to say, when this area of her back is percussed with the mouth open you will observe a peculiar note, which is known as the "cracked-pot" sound, and which is characteristic of a cavity which connects with a bronchus and so with the trachea and the outside air. It is not observed over the so-called closed cavities, where no connection with the outside air exists.

Now, the question which confronted us when we obtained these findings was whether the case was one of tuberculosis or whether it was an infection of the lung of some other type. During November she ran a temperature which was very variable. It was distinctly a septic temperature.

The remaining part of the case I can tell you about and we will excuse the patient. (Patient excused.)

In the first place, there is one characteristic thing in temperatures which takes place in tuberculous cases. A person who has pulmonary tuberculosis will have the maximum temperature of 100° to 101° F. between 1 and 4 P. M.—that is, rather early in the afternoon. In the morning the temperature is almost invariably subnormal, and in the evening the temperature begins to fall again, so that many of these patients have a temperature at 7 P. M. considerably lower than the temperature observed earlier in the afternoon. But in the early afternoon, between noon and 4 P. M., the temperature is high. This holds true not only in tuculosis of the lungs, but also in other varieties of tuberculosis. It seems as though various bacteria have certain distinguishing characteristics. For example, the pneumococcus betrays its presence by a mass of cold-sores. The tubercle bacillus is peculiar in that it produces an early afternoon temperature. The streptococcus usually produces a marked oscillation in temperature, a see-saw temperature—high in the afternoon or evening, and very low sometimes in the morning, with marked variations in the up-and-down swing.

Looking over this patient's temperature chart, we find that it does not accord with the ordinary range in cases of tuberculosis. For example, we might take the temperature here in this case on November 25th, which happens to be the page open before me at this time; I can take any other day as well.

At 6 A. M. the temperature was 100° F.; 9 A. M., 99° F.; at noon, 99.4° F.; 4 P. M., 100.2° F.; 6 P. M., 100.8° F.; 9 P. M., 100.8° F.

On the following day, on the 26th, the temperature at 6 A. M. was 99° F.; 9 A. M., 99° F.; at noon, 99.6° F.; at 3 P. M., 99.4° F.; at 6 P. M., 99.6° F.; at 9 P. M., 100.6° F.; at 12 P. M., 99.8° F. On both days the maximum temperature took place at night. Indeed, the same thing is true on the following day, her maximum temperature being 100.4° F. at 9 P. M. on that day. On the following day, that is to say, the 28th, her temperature reached its maximum (100.6° F.) again at 9 P. M.

Such a temperature range raises a presumption against the diagnosis of tuberculosis; it raises a presumption in favor of another kind of infection in the lung.

The case was, therefore, looked upon early in its residence here in the hospital as one not of tuberculosis; but the question which had to be decided, even if it were not tuberculosis and were a lung abscess, was as to how it could best be managed. Should it be managed on the expectant plan, which means waiting until ultimately death takes place, or should something be done to obviate death, which is almost sure to follow?

In order to come to some conclusion as to the operability of such a case one has to be very certain that the lung is fastened to the chest wall; otherwise, when one attempts to do an operation in a case of this sort one would run the risk of producing pulmonary collapse. The only way in which one can make certain that the lung is fastened to the chest wall by adherent pleurisy is to note whether the lower margin of the lung is movable or whether it is fastened. This can be done by percussion of the lower margin of the lung in deep inspiration and expiration.

In her case the mobility of the lower border of the lung on

the left side of the chest was excellent, and the mobility on the right side of the chest was so slight as to be unrecognizable. This meant that the right lung was probably stuck to the chest wall, but not that it was necessarily adherent. Since, however, normal resonance was found at the base of the right or diseased lung below the level of dulness, and since this line of resonance was not mobile, the presumption that the lung was adherent to the chest wall became very great.

Another question which had to be decided early was whether there was not a primary tuberculous infection of the lung, and whether it might not be possible that a secondary infection by another germ might have taken place and have produced a secondary abscess of the lung.

Going back in her history, however, one point is very evident, and that is that she was in a comparatively healthy condition as regards her lungs at the time of the operation for the removal of her tonsils on the 15th day of July. The only trouble she had ever had before that time was a slight cold in June, lasting for about a week. She was in a run-down condition, such as might have been caused from the intoxication of diseased tonsils; it was not a condition which would indicate the existence of tuberculosis of the lung.

We find next that the onset was sudden, comparatively speaking. One week after the removal of her tonsils she again took a cold, which "settled upon the lungs," as she expressed it. Such a cold settling upon one's lungs is often only ordinary bronchitis, and when we are told that in the month of August, just exactly a month after the removal of the tonsils, there was profuse expectoration of yellow sputum having a foul odor, we are driven to suppose that at that time the disturbance was not a tuberculous disturbance at all, but a putrid bronchitis—putrid bronchitis because of the foul odor which she clearly described.

Notwithstanding the fact that tubercle bacilli were reported by a physician as having been found in August, we must use our better judgment and say that he must have been mistaken, and that there were no tubercle bacilli, even though they were supposed to have been found. It is quite possible for one to make

mistakes in these cases. On many occasions errors have been made by using distilled water in which acid-fast bacteria have been growing. The individual who is preparing his slide must be sure that he is preparing it without contaminating it. At any rate, in this case we feel certain that there were no tubercle bacilli, notwithstanding they were reported present in the middle of August. In tuberculosis you have an insidious origin. In this case the onset was comparatively sudden. It is true that this patient was in a run-down condition, but it was because of the chronically diseased tonsils rather than disturbance of the lung.

Resuming the consideration of the history of this case, then, we find the sudden appearance of a putrid bronchitis and a comparatively high temperature; this lasted for a while in varying degrees of intensity; by September she had partially recovered, when there were a few days of normal temperature; then fever reappeared, and, finally, in October, suddenly there was a sharp pain beneath her right shoulder-blade with the spitting of blood, and she became very much worse.

What took place at that time? Probably there occurred at that time the infection of a vein in the right lung, or possibly, though not probably, arterial thrombosis; most likely a vein was directly infected by extension. Why? Because of the sudden rise of temperature and the sudden sharp pain beneath the right shoulder-blade. Such sudden elevations of temperature are rather characteristic of infections of veins and phlebitis.

It looks as though we had an individual who had previously been infected with a putrid bronchitis, who suddenly gets a new focus of infection, undoubtedly primarily venous, and this leads rather rapidly to the development of an abscess over a period of three or four days' time, so that within a short time she spits quite a large amount of sputum. The sputum is of the putrid type which she spat up in August, showing that there had been a reinfection by the same kind of germ which had infected her lung originally, and a germ, by the way, which is not infrequently to be found in the tonsillar tissue itself and from which it probably came; that is to say, we must look upon the tonsils as the

focus from which this trouble might well have sprung, coming on as it did so soon after the removal of the tonsils.

The next step in diagnosis, of course, is to put a needle into the chest to determine the point where the abscess is and whether it contains abundant pus. Remember that against the diagnosis of tuberculosis was the whole course of the case, the temperature range from day to day; the absence of tubercle bacilli, examined both in the ordinary way and by the antiformin method; the absence of disturbance in either pulmonary apex in the way of râles, prolongation of the expiratory murmur and dulness, and the presence of dulness low down in the chest, about opposite the seventh, eighth, and ninth ribs on the right side posteriorly. Remember, further, that this dulness was quite intense and that in the dull area bronchial breathing was to be heard, together with some large coarse râles.

Having these facts in mind, believing that the case is not tuberculous, but probably an abscess of the lung, the next step, of course, is the insertion of a needle into the supposed cavity for the purpose of seeing whether pus can be abstracted.

This was done and pus removed. One-third of a syringeful (using a syringe with a diameter of about $1\frac{1}{4}$ inches and 3 inches long) was removed. It was then determined to open the abscess at once. The patient was given gas, and Dr. J. B. Murphy, to whom I referred the case for operation, resected the eighth rib posteriorly and inserted a drainage-tube. The patient's condition immediately began to improve.

Previous to the operation there had been marked signs of sepsis. She had developed a very high range of temperature and was getting constantly worse. It was perfectly evident that such a desperate disease demanded a desperate remedy. At first she and other members of the family were rather unwilling to entertain the idea of an operation upon a supposed lung abscess, but her condition getting as it did so very much worse it became evident that something had to be done. For instance, here is the record of an ordinary day:

Pulse at 6 A. M., 124; temperature, 101° F.; at 9 A. M. pulse 120; temperature, 100° F. (that is, axillary, all of the tempera-

Fig. 3.—Abscess of right lung. Skiagram showing two-lobed cavity in right lung.

tures on this day being axillary); at 4 P. M. pulse not recorded, the temperature 101.4° F.; at 6 P. M. the pulse 140 and the temperature 103° F.; at 8 P. M. the pulse 146 and the temperature 103.4° F.; at 10 P. M. the pulse 134 and the temperature 101.4° F.

You can see what desperate straits the patient was in when you contemplate such high temperatures and pulse-rates. During that night she slept but four hours; she coughed a great deal and vomited a great deal; the coughing led to the vomiting. Her weight diminished to a great extent. Her sisters weigh about 140 pounds; she got down very low, we do not know how low her weight fell. After she had been operated upon she was weighed and her weight found to be 87 pounds. I think it not unlikely that her weight diminished even to 80 pounds.

She was quite gray in color; she was excessively weak; she was having night-sweats, and she looked like a patient in the third stage of tuberculosis—the end rapidly approaching.

She finally consented, as did the other members of the family, to the operation, but not until she had been in the hospital for over six weeks' time. The operation was done, and she has been here ever since, gradually getting very much better, as will be illustrated by the temperature record.

I would say furthermore that we had an *x*-ray made of her chest, and it shows evidences of a cavity situated where we had found it in our physical examination. The *x*-ray having been made and the chest having been punctured and the pus having been obtained, she was operated upon.

The temperature on the day of operation was 102° F., axillary; the pulse ranged from 120 to 124. The next day, the second day after the operation, for the first time in several weeks she had a normal temperature. Two days later her temperature at 6 A. M. was 98° F.; 9 A. M., 98.4° F.; at noon, 99° F.; 3 P. M., 99.2° F.; 6 P. M., 100° F.; and at 9 P. M., 100.6° F.; the pulse ranged from 120 to 126, and from that time on her condition improved very much.

The odor in her room was extremely foul, but now her room has no odor whatever. The odor from the drain was very bad

for a long period of time also, but that is now getting to be in good condition.

Her temperature has fallen so much that at the present time it has been normal for a period of eight days, and preceding this there have been only slight evidences of temperature. For instance, on the 1st day of March, 1915, she had 99° F. at 6 P. M.; she had on the 28th of February, 99° F. at 6 P. M.; on the 27th her temperature was normal; on the 26th, 99° F.; on the 25th, 24th, and 23d of February, and on the 22d, 21st, and 20th the temperature is recorded with only slight variation, the maximum being 99.2° F.

You will notice one thing which is extremely interesting—that is, as soon as the drainage was started at the bottom of this cavity through a tube, her temperature immediately fell. It fell the very next day, so that in a very short space of time her temperature was very much better, and with the falling of her temperature there was a falling of the pulse-rate.

The amount of discharge from the tube has grown markedly less. I think we can look for a complete cure as far as this abscess cavity is concerned. The drainage will have to go on for a very long time, until every particle of pus has drained out and until such time that no more pus is formed. When that takes place she will be practically well.

Of course, there will be a long period of convalescence during which fibrosis will take place in this lung. There will be, of course, a certain amount of insinking, which will be greatest over the rib removed. We hope that the process itself will entirely heal, and that she will be, to all intents and purposes, well.

She weighed 87 pounds a week ago and now $88\frac{1}{4}$ pounds; so she gained last week $1\frac{1}{4}$ pounds. She will be weighed from week to week, and I anticipate she will make a decided gain.

The coughing has not ceased because it is due partly to the irritation of the tube and also partly to the abscess, and also secondarily to pleurisy, which almost invariably takes place in these cases over the area of the diseased lungs.

This case is very important because there is a great tendency

to pass such cases by. The physician occasionally has such cases; they seem desperate, and he says to himself at times—if he has not a conscience—"Well, she is going to die anyway, and even if it is not tuberculosis, there is no chance; what is the use of doing anything?" That is the ordinary attitude which is taken. That is the attitude which is almost always taken—one of absolute dejection and discouragement—and yet, these cases don't have to die.

Some years ago when I was an attending physician in the Cook County Hospital I ran across a similar case. The man had, supposedly, a very bad case of tuberculosis. He was spitting up a great deal of sputum; he was running a high fever; he had severe night-sweats; he was extremely emaciated; in short, he had apparently not very long to live; he was a Scotchman and had lots of grit. On examination it was evident that he did not really have tuberculosis, but an abscess of the lung, possibly an interlobar empyema; he was operated on by one of the attending surgeons, and in the course of a few months made a complete recovery and left the hospital a well man. Had the diagnosis of tuberculosis been persisted in, he would have died, as many of these cases do.

When you find a case such as this patient, showing a history which is atypical in its origin, showing a possible infection of the lung by some other germ than the germ of tuberculosis, when the findings are not absolutely typical of tuberculosis, then think about the case, and think about it long and earnestly; think about the night-sweats, emaciation, cough and expectoration, and all the things that are ordinarily regarded as pathognomonic of tuberculosis. She had numerous hemorrhages, and yet there was no tuberculosis about it, as we are absolutely certain. The very fact that at the present time she has run eight days with absolutely normal temperature is proof sufficient that the process is not a tuberculous proposition. You cannot heal tuberculosis by the mere abstraction of pus in the short space of two months.

It requires a very long time to make any impression whatever upon a pus focus in tuberculosis, yet in this case drainage of about two months' time has sufficed to bring her temperature

nearly to normal, she having had no fever now for a period of eight days. The toxemia still remains; her heart is still rapid, reaching 100 at times, and falling, of course, to 86 and 88 at periods of great calm. It will be a long time before the myocardium will have recovered.

We do not have to make a bad prognosis so far as the heart is concerned. It is probably true that her life expectancy has been shortened by this disturbance, but it is not true that the myocardium is permanently weakened. Of course, this disturbance will have produced a marked effect upon her arterial system, and in the future her arteries will show degeneration before the arteries of her sisters show such degeneration. One cannot go through such a process without having some permanent effect upon the nutrition of the arterial walls, but it will probably not be early manifested.

There is one good thing in her case; that is, notwithstanding the immense amount of intoxication which she must have suffered, we have not found a disturbance of her kidneys; she was spared that trouble. Furthermore, she never got any joint disturbance. Having originally a tonsillar disturbance, we might have expected some joint disturbances, but she has been entirely free from them.

One other complication could have occurred. You probably are not aware of the relationship between a lung abscess and a cerebral abscess. Abscess of the lung, or empyema, is more apt to produce abscess of the brain secondarily than an abscess at any other place; or, one might put it thus, that abscesses of the brain are more common after abscesses of the lung or ordinary empyema than they are after any other kind of infection. So she might well have developed an abscess of the brain, but she didn't. She was very fortunate in that respect.

I very well remember the case of a physician with a sinus from an old empyema opening externally, who should have known of the relationship I have just mentioned. Because he did not fully appreciate it, it led to his death at the age of forty-three. He was making a call one evening and was suddenly seized with paresis on the left side of the body. The paresis was so sudden in

onset that he had to be put to bed in the home of the patient on whom he was calling, and remained there that night and the next day until he could be taken home. The hemiplegia deepened and fever followed. We localized an abscess in the subcortical motor area in the right side, because it had produced a hemiplegia instead of a monoplegia. At the postmortem examination an abscess was found beneath the right fissure of Rolando and extending deep into the medullary substance, so that it impinged upon the internal capsule in the anterior part of it. His death, therefore, was due to the sudden development of a cerebral abscess secondary to his old empyema imperfectly drained through a sinus opening in his chest wall. Of course, those possibilities are always present in cases of this type, and one having such a case as this always feels the possible risk of the patient to such a sudden termination.

We now feel perfectly easy about this patient. We feel that the disease is completely controlled. Now, we come back to the original cause of the whole trouble. We doubt not it was infection of the body from the removal of the tonsils. That does not mean that the tonsils must not be removed, but it means that they must be removed with the greatest care. Tonsils are apt to give rise to many disturbances if left to themselves, and at times it is imperative that they be removed. We are not finding fault with tonsillectomy. The tonsils lead to a vast number of cases of endocarditis, so-called rheumatism, and various joint diseases. If they are seriously diseased they ought to come out, but occasionally after the operation unfortunate complications may develop. You must not look upon tonsillectomy as a trivial matter. It is a thing to be done in a hospital, and should be done with sufficient anesthesia so that it may be done right and with no possibility of direct infection of the patient.

This patient's tonsils probably had to come out. She was unfortunate in that during the operation an infection took place, or that one took place subsequent to the operation. It is probable that the disturbance took place after the tonsils were removed from the tonsillar bed. It began as bronchitis, presumably early, then putrid bronchitis, and subsequently there

was the formation of an abscess, and, finally, the condition of extreme sepsis which she shows.

The case is one which carries a lesson. The message which it carries is this: Don't be cock-sure that a case is tuberculosis just because it has all of the superficial appearance of tuberculosis. Remember that the diagnosis of tuberculosis is oftentimes made when it ought not to be made; just as oftentimes it is omitted when it should be made.

There is no medical treatment for abscess of the lung. The treatment can only be surgical. You may give medicine to these people as long as they live. They will die still taking medicine. If, however, you can get in where the pus is being made and drain it from the bottom you can effect a cure. The reason no cure by medicine can be effected is that no drainage can take place. Such coughing as there is succeeds only in taking the "cream" off the pus-mass. That which lies beneath remains there. The only way is to get a tube into the bottom of the abscess and then the pus will run out and the case will heal.

So we do not speak of medical treatment in cases of this sort.

[NOTE.—May 9, 1915: The patient's condition is satisfactory, but the result is not brilliant. The weight went up only to 92 pounds, and now it has fallen back to 90 pounds. The reason for the continued expectoration was thought to be a lack of communication from the chest wall through the abscess cavity to the trachea. Methylene-blue solution put into the tube was coughed up by mouth in ten minutes. Lately, on two occasions, sequestra of lung tissue have been coughed up. Until such time as all lung sequestra are done away with, fever will follow and expectoration will be profuse. The patient's appetite remains fairly good. There is still much cough, and at times there is some fever, the degree of temperature standing in close relation with the amount of drainage.]

A LESION OF THE CAUDA EQUINA; SCIATICA THE MOST PROMINENT SYMPTOM

INTERN'S HISTORY: The patient is a male, forty-six years of age, a railway engineer by occupation. He entered the hospital February 27, 1915, in another service, having been in the hospital previously, in December, 1914. At his entrance he complained of pain in his right thigh, the pain starting at the sacrum and running down the back of the thigh to the calf of the leg. The pain is not present while the patient is lying down; it is present when he is sitting down, and especially on rising after he has been sitting for some time. It occasionally comes on while he is walking. He also complains of pain in the *left* knee when he walks up steps. He can flex the left knee without pain, but if weight is put upon it and he flexes it on going up steps it pains him. The left knee has never been red or swollen.

The duration of the pain in the right and left legs has been for two years. He sits in his cab with the weight thrown over to the right side, and states that his engine is a "rough rider." He has never had any previous illnesses; he has never met with any accidents; he has never had any infections of any sort; no sore throat and no nasal sinus disease. He feels perfectly well except for the pains in his right leg and occasionally in his left knee.

The family history is negative; there is no history of malignancy. It is true that his father died of tuberculosis, but the patient himself has always been robust. He had a Neisserian infection at the age of twenty-five followed by a bubo; he denies ever having had a luetic infection.

DR. MIX: As you have heard, the patient is an engineer, forty-six years of age. He has had pain in the right thigh of so severe a degree ever since last July that it has been impossible for him to do any work at all. His pain has been so severe that he has been more than anxious to have an exploratory laminectomy done to see if something might not be found to account for the pain or to remove it. The pain which he complains of is not like that which is found in an ordinary sciatica. When the

patient entered the hospital he came believing that he was troubled with sciatica, since that diagnosis had always been made in his case. You will note, however, that his "sciatica" does not involve merely the right leg, although it is true that the sciatica which came on in July, 1914, affected, for the most part, the right leg with the greatest severity. It is also true that he has had pain in the *left* leg in the region of the knee which is not associated with signs of an arthritis. It is a question, therefore, as to whether he has a simple sciatica or whether there is something else the matter with him. A great many cases are turned off by physicians with a diagnosis of sciatica because they do not take the trouble to study their patients. Sciatica is nothing but a symptom. If a man has pain in his leg he has some reason for it, and the reason is not ordinarily an involvement of the sheath of the sciatic nerve. The probabilities are that in this patient's case we shall be able to arrive at some other diagnosis than a simple sciatica.

Whenever you see a patient complaining of sciatica in one leg, with a history of transitory pain not easily explained occurring in the other leg, the probabilities are extremely strong that either the spinal column is involved or there is some inflammation or pathologic condition affecting the cauda equina or the sacral roots. Many cases of double sciatica are really due to a spondylitis involving the two lower lumbar vertebræ. Possibly the most common cause of all cases of sciatica is spondylitis either of the last two lumbar vertebræ or of all of the lumbar vertebræ. Frequently such cases of sciatica develop in the fall of the year. They often come to Mercy Hospital in the season when bronchitis and respiratory troubles are beginning to develop. Ordinarily in these cases there is a history of an acute onset, with backache of considerable severity lasting for a few days, and then the subsequent development of what has been called rheumatism or sciatica.

Others of these cases of so-called sciatica are due to involvement of the spinal roots in the spinal canal by some sort of an inflammatory process. The present stage of our knowledge in regard to lesions in the cauda equina is one of only partial en-

lightenment. As more and more operations are being done on the spinal canal more living pathology is coming to light in regard to the cauda. In the May, 1914, number of the American Journal of Medical Sciences a hitherto undescribed disease of the cauda is reported by Kennedy, Elsberg, and Lambert, in which an intense inflammation of the roots of the cauda was found in 5 cases upon performing an exploratory laminectomy. On account of the fact that this man's sciatica is bilateral, even though it be predominantly on the right side, it is necessary for us to examine his nervous system carefully.

Before beginning the examination of this patient I wish to state one thing rather emphatically, and that is, that cauda equina lesions, usually a frequent cause of symptomatic sciatica, are almost never bilateral with an equal involvement of the roots on the right and left sides. It always happens that some roots escape on one side or the other. In these cases, therefore, we never find a bilateral involvement which is exactly the same on both sides. This is a diagnostic point of very great value and applies in this man's case, for you will remember that the great bulk of his sciatica is on the right side. He has very little pain in his left leg.

DR. MIX (to Patient): When did you first have pains in your right leg?

PATIENT: About one and a half years ago.

DR. MIX: Where did they begin?

PATIENT: On the outside of the thigh.

DR. MIX: That is, then, in the region of the supply of the external cutaneous nerve. The so-called meralgia paresthetica which involves the territory of supply of the external cutaneous nerve is sometimes due to a lesion in the spinal canal.

DR. MIX (to Patient): Where did you next notice the pain?

PATIENT: There in the notch, a painful catching.

DR. MIX: What kind of a pain was it?

PATIENT: It was pretty bad; it would almost take my breath away.

DR. MIX: Was it sudden?

PATIENT: Yes.

DR. MIX: Was it stabbing?

PATIENT: Yes.

DR. MIX: Then it would go away and you would be all right for a while?

PATIENT: Yes.

DR. MIX: What was the next thing you noticed?

PATIENT: It began to hurt me right there (pointing).

DR. MIX: At the level of the synchondrosis on the right side. He says that it would hurt him while he was sitting or while standing, but not while he was walking about.

DR. MIX (to Patient): When you lie down is it better or worse?

PATIENT: It is not so bad lying down, but when I sit I notice it more.

DR. MIX: What was the next thing you noticed?

PATIENT: About in July the extreme end of my spine became sore to the touch.

DR. MIX: What was the next thing after July?

PATIENT: I just suffered from that pain.

DR. MIX: Did you have any pain in the left leg?

PATIENT: When I hold the right leg straight up it hurts the left leg.

DR. MIX: Have you had any other disturbances similar to this?

PATIENT: Yes, I had a rheumatism that left a kind of a numbness behind it probably twenty-five years ago.

DR. MIX: Have you noticed any loss of power in either of your legs?

PATIENT: When I was in the hospital about the middle of December I noticed I could not bend the left knee very well.

DR. MIX: When did you notice that?

PATIENT: About the 23d of December; when I would step off with that leg the knee would bend down.

DR. MIX: The fact that his knee gave way indicates a disturbance in the nerve supply of the anterior crural nerve. The quadriceps extensor muscle, which prevents the knee from giving way, as the patient expresses it, is supplied by the anterior

crural nerve which comes from the second, third, and fourth lumbar roots of the spinal cord. You will note that his disturbance in the right side began in the territory of the external cutaneous nerve which comes from the second lumbar segment of the cord. In other words, the disturbance on the right side and the disturbance on the left side both reach as high as the second lumbar segment of the cord; or, putting it another way, as high as the second lumbar roots on both the right and left sides.

You will also note that the disturbance on the right side began as a sensory disturbance in the form of a neuralgia of his external cutaneous nerve, and that the disturbance on the left side began as a motor disturbance with a giving way of the left knee when he tried to go up steps, the onset being approximately the 23d of December. These facts are of great importance in arriving at a conclusion as to a disturbance in his spinal canal.

In examining the patient we will start with his knee-jerks. There were none in December and there are none present now. We do not know when he lost his knee-jerks, but we do know that they are absent. The loss of the knee-jerks means a disturbance in his reflex arc. Either there is a defect in the passage of the sensory impulse to the cord or there is a defect in the cord itself. Since he is able to use his quadriceps extensor muscles we are quite positive there is no defect in the return impulse through the motor nerves. Knee-jerks are lost in cases of multiple neuritis, or locomotor ataxia, or pernicious anemia with involvement of the columns of Goll and Burdach. The loss of the knee-jerks in general usually means either some disturbance or disorder in the posterior columns of the cord, or else some disturbance in the peripheral nerves, particularly the sensory ones. We must ask ourselves in the present case whether we believe the lesion lies in the posterior columns of Goll and Burdach, or whether it lies in the anterior horn cells, or whether it lies in the sensory root. We can answer the question as to the likelihood of its involving the anterior horn cells at once. Inasmuch as there is no atrophy in the quadriceps extensor muscles we may be certain that there is no involvement of the anterior horn cells.

Now let us examine the ankle-jerks. On having the patient kneel on a chair, with his feet out behind him and striking the Achilles tendons, we find that there is no response in either foot. The ankle-jerks are absent. The fact that both the ankle-jerks and the knee-jerks are absent is a positive indication of some organic disease somewhere in the reflex arc.

We will next test for the Babinski reflex. We would expect it to be absent in such a case, because ordinarily if it is present the knee-jerks are increased and there is ankle-clonus. You will notice that as I stroke the sole of the foot the big toe turns down both on the left and on the right foot. In other words, the plantar reflex is a perfectly normal one, and the Babinski toe-sign is not present. We also find that there is no ankle-clonus. Inasmuch, then, as both the patellar and ankle-jerks are absent, and inasmuch as there is no Babinski toe-sign, no ankle-clonus, no spasticity, we may be certain that there is no involvement of his pyramidal tracts.

The patient is a man forty-six years of age—the tabetic age. May he not have tabes? The knee- and ankle-jerks are absent, as they are notably in cases of locomotor ataxia. Has he become a victim of that disease? Not at all. In the first place, there is a negative Wassermann, not only of the blood, but also of the cerebrospinal fluid which I removed and had examined when I first saw him in December. Furthermore, not only was the Wassermann test negative, but there was no evidence of involvement of the meninges in December when I made my spinal puncture. Not many cases of locomotor ataxia exist with a negative Wassermann reaction on both the blood and cerebrospinal fluid, without the presence of Lange's colloid gold reaction, or with no increase of the lymphocytes on examining the cerebrospinal fluid. Moreover, in the case of this man there is no history whatever of luetic infection. True, he had a Neisserian infection when he was twenty-five years of age, but he is the father of a very healthy set of children; his wife has had no miscarriages, and he positively asserts that he never had a luetic infection. Furthermore, when we examine the eyes for the Argyll-Robertson pupil, so characteristic of tabes, we find that it is not

present. The pupils react perfectly to light and in accommodation. There are no gastric crises, no delayed sensation, no Romberg sign, no ataxia, no ptosis of either eyelid, no paralysis of the recti muscles of the eye. It is true that he has had some sudden, shooting pains, particularly in his right leg, slightly in his left, which resemble the lightning or shooting pains of tabes, but we believe them to be due to another cause.

The principal facts which we have thus far brought out are the motor disturbances in the left leg, the excessively severe pains in the right leg, and the absence of knee- and ankle-jerks. Let us next examine the abdomen. What is the condition of the abdominal reflexes? Going back to the absent knee-jerks, we were led to a conclusion that there might be an involvement either of the columns of Goll and Burdach, such as occurs in tabes with absent knee-jerks, or a disturbance of the posterior nerve-roots by reason of some lesion of the cauda equina. Multiple sclerosis might be indicated by the motor disturbance in the left leg if these abdominal reflexes are not present, but multiple sclerosis is almost never associated with a loss of the knee-jerks and the presence of pain. On the contrary, it is usually associated with increased knee-jerks, the Babinski toe-phenomenon, and the entire absence of sensory phenomena. When we come to test his abdominal and epigastric reflexes we find that they are present on both sides.

Is there any way in which we may be able to test out the possible involvement of the columns of Goll and Burdach? Yes. We will ask the patient to close his eyes while we slowly move first his right and then his left big toe. He is to tell us in which way we move the toes. You will notice from his replies that he has exact knowledge. There is, therefore, no loss of his muscle- and joint-sense. In tabes the loss of the muscle- and joint-sense shows up very early, so that even in beginning cases this test which we have just shown you is found positive. Since the patient does not lack knowledge as to the direction in which his big toes are moved, it is evident that there is no involvement of the columns of Goll and Burdach. Of course, what I have just said would not be true unless we ruled out the possibility of entire

destruction of his posterior nerve-roots. This we have already done, however, though we have not before said so, because we have tested his tactile sense over the lower extremities, both right and left, and find that there is no loss whatever.

Before turning to the motor phenomena it may be well to call your attention again to certain peculiarities in regard to his pain. Curiously enough, the pain is not present when the patient is lying down. It comes on when he sits or stands. I have also found, by inquiry, that it is especially severe when he sneezes or coughs. The presence of pain in the lower part of his back and in the region of the sciatic nerves on sneezing or coughing ofttimes indicates a disturbance in the intraspinal canal. It is a notable symptom in many tumors of the spinal cord involving the region of the cauda equina. If any of you have done a lumbar puncture you know that if the patient coughs while the needle is inserted there is a spurt of cerebrospinal fluid, and it is easy to see why sneezing or coughing in this case produces the manifestation of pain.

Remember that nothing has even been found in this man's legs to account for any local disturbance in the sciatic nerves. Indeed, we are at once justified in eliminating the possibility of sciatica. There is no particular pain over the trunk of the sciatic either at the sacrosciatic notch or at the bifurcation of the sciatic nerve, or where the external perineal nerve passes around the head of the fibula. I can roll this man's external peroneal nerve under my finger, as you see, on both the right and left sides, and it gives him no particular pain. Moreover, when I extend this man's leg to the utmost there is no increase in his pain such as there would be if he were suffering from sciatica. He can lie in bed and stick his foot straight up in the air and move it in any direction without pain. When he sits up, however, the pain comes on. Clearly, a pain behaving as this man's pain behaves is not ordinary sciatica.

Further examination shows no sensory disturbance in the way of an anesthesia, hyperesthesia, analgesia, hypalgesia, or thermanesthesia. Neither have there been any bladder or rectal disturbances. His sphincters are under perfect control.

Is there any atrophy in his legs? We have heard of the subjective weakness in his left knee which occasionally drops him. Is there any atrophy? The two legs look exactly alike, and so far as I am able to observe there is no atrophy in the legs below the knees. When we come to examine the thighs they look to me to be about of the same size. Now let us ask ourselves a question: Suppose only a few nerve-roots are involved, what would be the effect? The effect would be that only a few muscle-fibers would be involved, and it is possible that so few of them might be involved that even if atrophy were present in them we would not be able to detect it. We cannot, then, in this case satisfy ourselves either that there is no disturbance in his anterior horn cells or his lower motor neurons. All we can say is that he is weak in his left knee and that he has pain in his right leg.

We are now in a position to discuss the question of diagnosis. There is nothing to make one believe that there is a pachymeningitis internus, though the possibility exists from the cerebrospinal fluid which was obtained. I neglected to state that a lumbar puncture was done yesterday, and that it showed ten white cells to the field in a speicmen not centrifuged. A normal uncentrifuged specimen ought not to show more than two to four cells to the field. The very slight increase of the lymphocytes is indicative of a slight disturbance of the meninges of the cord.

Is there any reason to believe that he might have a luetic infection of his spinal meninges? This question we must answer in the negative because of the negative Wassermann reactions on both blood and cerebrospinal fluid, and the negative physical examination. Remember that a lumbar puncture was done in December, and on that occasion the cerebrospinal fluid was negative when tested by the Wassermann test, and that the blood was also negative in December to the Wassermann test. Further, recall that a second puncture done recently was again negative, and that the blood when again examined was found negative. In the presence of these negative findings, in the absence of an abnormal obstetric history, and in the absence of luetic history or infection in all his forty-six years of life, we are forced

to the conclusion that he cannot be suffering from syphilis of the cord.

Is it possible that he has locomotor ataxia? No. The reason for that answer is that locomotor ataxia is invariably caused by syphilis, which this man has never had, to the best of our knowledge and belief. The earliest manifestations of tabes are the shooting pains, the loss of the muscle- and joint-sense, the gastric crises, and the girdle sensation; subsequently sensory disturbances appear, especially slight loss of the sense of touch around the shin-bones, ankles, and calves. None of these symptoms have occurred in this man's case, with the possible exception of the shooting pains in his right leg.

Is the lesion a multiple neuritis? No; because there are no sensory disturbances of touch and of temperature and of pain, because there is no pain on pressure along the nerve trunks, and because there is no atrophy and no loss of muscular power except the slight loss in the left quadriceps extensor. The diagnosis of multiple neuritis, moreover, ought never to be made unless an etiologic diagnosis can be established. So far as we are able to find out, this man has never had any cause capable of giving rise to a multiple neuritis, either infectious or toxemic.

Why do we think the lesion is a caudal lesion? Because the knee-jerks and ankle-jerks are gone. If the lesion was in the spinal cord there would be increase in the knee-jerks, with probably the Babinski toe-sign and possibly ankle-clonus. In this case none of these phenomena are present, and the lesion must lie, if a lesion is present at all, at or in the cauda equina.

What must be the nature of the lesion? That must be conjectured. It might be a neoplasm, a fibroma, or an osteoma. It might even be a tuberculoma, although we do not think it is in this case, because there never have been any symptoms indicating tuberculosis of the bone. It might be an inflammatory affair; the chances of its being so are present because of the increase in the white cells in the last spinal puncture. Indeed, it is more likely that the lesion is inflammatory than that it is neoplastic. We must remember, however, that, like many

cases of tumor, the first thing this man complained of was pain, and it was a long time before symptoms of a motor sort appeared; then they appeared in the opposite leg. Such a state of affairs is possibly more likely to occur in cases of neoplasm than in cases of inflammation.

Against the idea that the disease is one affecting the cauda, the absence of bladder symptoms and rectal symptoms might be urged. In reply to this objection I might, however, say that every case of lesion of the cauda equina that has ever existed had a stage previous to the stage of involvement of the bladder and of the rectum. Is not this man in that stage? What sense is there in waiting for the manifestations of the disturbance of the sphincters of the bladder and rectum before we attempt to make a diagnosis? We know that his knee-jerks are gone, that his ankle-jerks are gone, that he has definite root pains in the right sciatic territory, and that he has a motor defect in the left anterior crural nerve territory. We note that the level of involvement on the two sides is approximately the same, pain being experienced in the territory of the external cutaneous nerve on the right side, and motor defect in the anterior crural nerve territory of the left. To be sure, he has no sensory disturbances. We admit that there is no saddle-shaped anesthesia which is usually demanded by most diagnosticians as a necessary criterion for the diagnosis of caudal lesions. Again we ask, Is it necessary to wait for a patient to develop such anesthesia before one makes the diagnosis of a caudal lesion? Have we not sufficient data present already to justify the conclusion that he is suffering from an intraspinal caudal lesion? My own answer to this question is decidedly in the affirmative.

All of these possibilities as to diagnosis have been placed before the patient. We have told him that we are in favor of an exploratory laminectomy, and we have asked the patient whether he wants it, and he says that he does. It is, therefore, going to be done. I believe the time is coming for exploratory laminectomies to be made, and I believe that when we come thoroughly to a realization that in a given case the trouble is an intraspinal affair, if we cannot make a positive diagnosis we ought

to have the spinal canal opened. It is hard work for the surgeon, of course, but striking results may sometimes be obtained. You will find in a great many text-books that there is a belief expressed on the part of the men who write these books that the spinal canal should not be opened in the case of caudal lesions until the diagnosis is made doubly sure by the presence of saddle-shaped anesthesia and the presence of bladder disturbances. What is the use of waiting until the pathologic process has become so severe in the region of the cauda equina as to produce destructive lesions in the third and fourth sacral nerves which supply the bladder?

It may be possible that in this case nothing at all will be found when a laminectomy is done. That may very well happen. It sometimes is true that only the microscope will show a lesion. It may be so in this man's case. We hope, for the patient's sake, that the pathology which is found will be macroscopic rather than microscopic, and will be capable of removal. A laminectomy will soon be performed in his case, and we shall be able to inform you subsequently about what is found. It is better to see these cases before operation than afterward. Now that you have seen the patient before anything has been done, when you afterward learn the findings you will be able to derive more positive instruction from his case.

[Note.—A laminectomy was performed by Dr. John B. Murphy at Mercy Hospital upon this patient on the 8th of April. Dr. Murphy made his incision over the spinous processes of the eleventh and twelfth dorsal and first and second lumbar vertebræ. He first clearly located the twelfth dorsal vertebra and then the first and second lumbar spinous processes. The operation of laminectomy was done in the usual way, the spinous processes of the vertebræ first being clipped off and then the laminæ removed. Owing to the fact that the man was very heavy the operation was rather difficult, the wound being so deep that the deep retractor did not reach the bottom of the field. A good deal of bleeding took place, necessitating packing until the bleeding was controlled. In a few moments the field was made perfectly dry by the compression of the sponges.

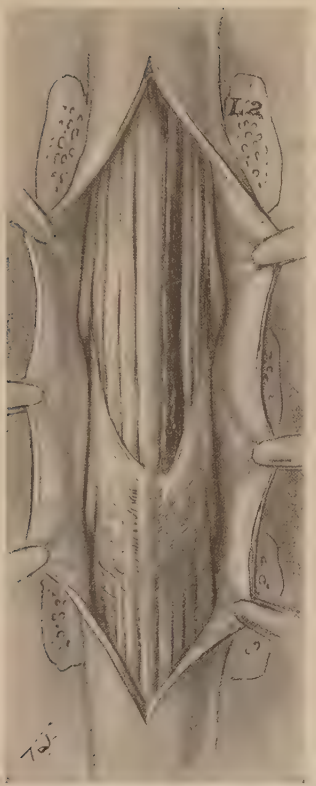

Fig. 4.—Veil of leptomeningitic thickening as a dense band at the top, gradually thinning out 1½ inches below. Note the very slight depression across the cauda ½ inch above the upper level of the veil. This veil matted together and bound down various of the caudal roots.

The dura was next fully exposed. It lay very deep in the field, being almost 4 inches from the surface. Across the dura covering the cauda there seemed to be an indentation caused by the bone protruding at that point and compressing it slightly. (Note the indentation in Fig. 4.) Even after the bone was removed the slight binding of the dura was present. At this point Dr. Murphy was unable to state whether the cause of the patient's pain was due to this fact or to something else. Before the dura was opened the bleeding was entirely stopped by the use of Horsley's sponges.

Next the dura was incised. Immediately the cerebrospinal fluid escaped in fairly large quantity. At once a local lepto-meningitis was disclosed with a dense webbing across. On testing with the faradic current the spinal root transmission was found to be perfect. This webbing which bound down the cauda was cut and the binding of the cauda released. Subsequently the dura was approximated and accurately sewed and the wound closed in the usual way. Figure 4 shows, as closely as it can be made to do so by the artist, the condition which was found at the cauda at the time of operation.

Dr. Murphy's comments at the time are quoted, "There was a cicatricial connective-tissue webbing which passed across the cauda from one side to the other, forming and involving one strand in the middle. This strand was found to pass out through the fourth lumbar root. This strand in appearance was cicatricial and differed from the other strands. There was no evidence of a neuritis of the caudal roots. Just above this connective-tissue mass was a depression in the cauda which was recognized before the dura was opened and remained present on the caudal filaments after the dura was severed, showing that there had been a continued pressure there. Whether that compression was the cause of this meningitic connective-tissue formation we do not know, but we must make the records so that in the analysis of these cases at any time in future years some observer may be able to arrive at a conclusion. I would say, however, that the compression in the dura did not seem to be the cause of the cicatricial tissue because the cicatricial tissue

was not confined to the particular spot in which the sign of compression existed. Just what rôle the white cicatricial strand which passed into the right fourth lumbar root plays in the symptomatology of the case I do not know. The filament was markedly cicatricial. The leptomeningitis which covered over the cauda in appearance was rather delicate and gelatinous-looking, though, in reality, very firm and strong, and spread over the cauda for nearly an inch. The appearance of this tissue was not unlike the appearance of the placental cord. A funis looks rather fragile, but when you come to pull upon it you find that it is exceedingly strong. This connective tissue covering the cauda looked rather transparent and slightly gelatinous, but was really also very strong, very firm, and unyielding."]

MAY 5, 1915: The patient since the 8th of April has been getting steadily better. He has had no sciatica since the operation. His power in both legs as he lies in bed seems perfectly normal; no sensory disturbances at any time have appeared. The only complaint which the patient has made is in regard to the soreness in his back from the laminectomy wound. This is getting better every day, the wound having healed by first intention, and it is likely that the patient will soon be able to return to his home and to his work as an engineer.

JUNE 7, 1915: The patient has continued to improve since May 5, 1915, and today he is leaving the hospital. His back is entirely healed, the pain has completely disappeared, and all that remains at the present time is a slight subjective feeling of weakness in the knees.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

NEPHRITIS. THE CONTRACTED KIDNEY WITH CARDIAC DECOMPENSATION. SIGNIFICANCE OF BLOOD-PRESSURE CHANGES.

I WANT to show you today some cases of nephritis, and discuss with you some of the different forms, particularly as we see them here in the County Hospital. I will read the salient points in the history of the first case:

This man, a teamster, twenty-six years of age, single, an American, came in on the ninth of this month. He had five complaints—first, dyspnea; second, swollen legs; third, nosebleed, which, as it came from a trauma, we needn't consider; fourth, chills; fifth, pain in the limbs and cough. This is his first attack of such an illness; was taken sick about the 2d of March, at which time he began to experience pain in the feet lasting a few minutes. This was worse when he got up in the morning and was aggravated on walking. The next symptom was swelling of the feet and legs. This never subsided entirely and was severe enough to make work impossible. Dyspnea was continuous, occurring even while lying down, was aggravated by walking, and was relieved, but not entirely, by a powder which was prescribed for him. Chill occurred at about the same time, lasting all night, and was severe enough to make his teeth chatter. No recurrence of the chill. For the last few days he has had a short, hacking cough, worse at night. When we sum up these points in the case we find it very simple—not many complaints.

I am not going through the entire examination with you, but I will read the salient points because I want to contrast this case with another one. It is as typical a case as you will

ever see. Neither the upper nor the lower eyelid is actually edematous, although both look a little puffy. There is no edema of the chest or abdomen, but when we come to the lower extremities we see that they are very edematous—I can push the finger-tip in fully an inch, and the edema is fresh. We know this by its softness; it is always fresh when it is of a "squashy" consistence; in an older stage it is always more firm and solid. Outside of the slight puffiness of his eyes we find only the leg edema on examination. Physical examination shows nothing abnormal except a few râles on auscultation over the lungs. Now, as regards the heart, to which we want to give very especial attention, we find that the apex-beat is not visible; it is, however, palpable in the fifth intercostal space just within the nipple line, and is a strongly heaving, well-defined apex-beat. I need not tell you that a sharp, circumscribed, heaving apex-beat points to a hypertrophy of the left ventricle. *The tones are all clear*, and the second aortic sound is enormously accentuated, from which we are justified in assuming one of two things—either that the aortic valves are sclerotic or that the patient has an increased blood-pressure. When last taken the pressure was 240 systolic and about 180 diastolic.

On examination of the abdomen the only noticeable thing is that when we come up over the upper part of the abdomen we find that the liver is swollen and tender and the edge can be felt with great ease within ½ inch of the navel. Now, examination of the urine shows the following points, which I will ask you to bear carefully in mind: In the first place, it is amber in color, specific gravity 1009, acid in reaction, a small amount of albumin, no sugar, a few blood-cells, a few leukocytes, and some hyaline and fatty casts. What I want to say about this particular patient is this: He comes in with one thing perfectly established—he has an edema, and the nature of that edema is mechanical, developing in the dependent parts first. That edema is a cardiac edema because it is mechanical, and associated with it is a large, tender, and swollen liver, the typical picture of cardiac breakdown. We would not have any suspicion that it was anything but a cardiac case except that we find

albumin in the urine and this enormously increased blood-pressure. It is evidently not a valve lesion, but one of the secondary dilatations of the heart, and in this case secondary to interstitial nephritis. The thing I want to impress on you is this: the majority of cases of interstitial nephritis come in with a broken-down heart. They do not come in with the picture of a nephritis *per se*. They come in with a cardiac picture, and if you are not careful you will overlook the kidney component entirely.

Here we have a young fellow of twenty-six, a hard drinker—a gallon of beer a day and whisky with it. Let us suppose now the patient gets a contracted kidney; he gets it very slowly if it is a primary contracted kidney, or he gets it less slowly if it is a secondary contracted kidney. The etiology of the nephritis in this case is almost certainly just straight alcohol. The man, through his enormous consumption of malt liquors with a certain amount of whisky on top, developed a chronic diffuse nephritis, and from this the secondary contracted kidney has resulted. With this chronic diffuse nephritis the blood-pressure became higher, and went up until we find it 200 or more as the interstitial changes become greater. As the sclerotic process has advanced the amount of albumin has become less, and the patient has gone on that way for a long time.

The process, then, in a case like this is the development of a chronic diffuse nephritis through the enormous consumption of alcohol. Then the secondary contracted kidney develops on top of the diffuse nephritis, and it goes along that way for a few years until the heart breaks down. That is why the great majority of cases that come into our hospital come with broken-down hearts. This patient has become short of breath and has a cough due to the venous congestion of the lung. Objectively, we find the clean-cut picture of cardiac insufficiency—a little congestion of the lungs, a swollen and tender liver, and edema of the legs. The albumin is increased in amount because there is venous congestion of the kidneys. This does *not* mean *acute nephritis*, but simply that severe decompensation with venous congestion of the kidneys has set in.

Most of these cases that come into the hospital are diagnosed as acute nephritis. Let me make this statement to you: In the wards of a general hospital like the County, acute nephritis is not a very common occurrence, and if we exclude the cases that follow the acute infections, such as those following scarlet fever, etc., a pure, acute nephritis is a fairly rare thing in a hospital like this. One could make out a pretty good argument for this being a case of acute nephritis. The patient is young, looks healthy, and was taken sick only four or five days ago. You may say, what more would you want for an acute nephritis? Now, as a matter of fact, the dropsy is a clean-cut cardiac dropsy; the nephritis is a clean-cut chronic interstitial nephritis, following a diffuse nephritis. The causation of diffuse nephritis frequently lies in whisky and beer, and in this case they are about equally potent. Remember, in a man who drinks hard the disease may develop at a pretty early age—he's only twenty-six. Again, when such cases come under observation it is usually because of cardiac breakdown. Lastly, when you find a man with a blood-pressure of 240 it can't by any possible stretch of the imagination be a new, a true acute nephritis. That he may have a slight, mild attack of acute nephritis on top of a chronic nephritis is barely possible. If it is a new nephritis the prognosis is pretty good; if he has an old nephritis with a contracted kidney the prognosis is very different, practically always fatal.

In all these cases, of course, we do a functional kidney test; this case was injected with phenolsulphonephthalein at 9: 22, and the first color came through the kidneys at 12:40. The normal time, as you know, is anywhere from fifteen to twenty or thirty minutes; since this took so long we were not able to finish the examination, but I may be able to tell you before we leave the clinic. This phenolsulphonephthalein test is very simple to make: The patient is given two or three glasses of water; then the bladder is emptied, and 1 c.c. of the solution of phenolsulphonephthalein is injected deep into the lumbar muscles. The catheter is slipped into the bladder and the urine allowed to drain into a test-tube containing two drops of KOH. The first drop which shows a pinkish color will give the first evidence of the phthalein. Now

withdraw the catheter and at the end of one hour you collect the urine, that is, one hour from the time of the first appearance of the phthalein in the urine. Then you collect for the second hour —take the urine again one hour after the first. The outputs should be something like this: In the first hour after appearance you get somewhere from 40 to 60 per cent. of the total amount injected. In the second hour, 20 to 30 per cent. So if we take an average case we find that about 60 to 70 per cent. of the phenolsulphonephthalein comes through in the first two hours. They have just finished the phthalein test on this patient, and the results are—first hour, 22 per cent.; second hour, 15 per cent.—which is, of course, very much below the normal.

Here is a second patient with a similar condition. Man aged thirty, laborer, Polish. Came to the hospital three months ago with the same complaint, which came on in the same manner as we have described above. Entered three months ago with attacks similar to this and went out again in a short time, much improved. Swelling of the feet and legs, not great enough to prevent work, but gradually becoming worse and extending until it reached the scrotum and abdominal wall, is his principal complaint. Then the back became involved in the edema. With this he had a cough, rather a moist productive cough with formation of a glairy mucus. He has now developed dyspnea, although this symptom has not been very marked. He did not mention this, but when we questioned him we found that he recognized himself to be distinctly short of breath. I want you to notice the curious, turgid facies—"bloated" comes nearer being the expression than anything I know of—but you will see that there is no edema in the face worth mentioning. If you saw him on the street you would probably say, "There is a typical old County Hospital 'soaker'." Now let us look at the edema of which he complains—mind you, he has been in bed some time, and so it has gone down very materially—we can see that the legs pit $\frac{1}{2}$ inch, and you can see that this edema is hard and takes some force to pit it, so we can conclude that it is an old, solid edema and has been there much longer than the one I have just shown you in the first case.

The abdominal wall is decidedly edematous; the scrotum and penis were greatly swollen, but the latter swelling has gone down somewhat. The back is most edematous of all; you can make a walnut disappear in the impression I am making in his back. *Again a typical mechnical edema*—it has disappeared from his feet to a large extent, owing to his being in bed, and has developed in his back—a typical cardiac edema.

On examination of his abdomen we find the liver down within 1 inch of the navel. You can see how he winces when I make pressure over the liver, but not a bit when I go lower. This is a duplicate case to the last one, so far as that point is concerned. Now we hear further that he rises two or three times at night to urinate; this has been going on for two or three months; previous to that time he never got up at night. When we examined the urine we found that it had an amber color, specific gravity of 1012 to 1013, with a fair amount of albumin constantly present, with a few blood-cells, hyaline and fine granular casts. No sugar. The twenty-four-hour quantity was considerably diminished. The phenolsulphonephthalein test on him was as follows: He was injected at 10: 23, and the first came through at 10: 35; in the first hour 25 per cent. had appeared, and in the second, 18 per cent., so there is a very decided decrease in the functional power of his kidney.

The blood-pressure is 156 systolic and 120 diastolic. Just a word or two about the blood-pressure—probably the most important thing we have to consider in nephritis. The man has an outspoken picture of cardiac failure, dyspnea, engorgement of the liver, dropsy of the feet, and dependent parts—in short, an outspoken mechanical dropsy from cardiac insufficiency. He now has a blood-pressure of 150 in spite of a broken-down heart. Had his heart not broken down his blood-pressure would undoubtedly have been much higher, possibly twenty to thirty points higher. It was probably at least 170 or 175 before he was taken sick. Now, gentlemen, please notice that in this patient we have chronic nephritis, coming in with the picture of a broken-down heart. He came in not because of the *contracted kidney itself*, but because of the superimposed cardiac condition. Every-

body who has been in private practice knows that these patients often come to us for the relief of symptoms dependent on the kidney—such as headache, impaired vision, dyspeptic symptoms, etc. The majority of our hospital cases come to us with the picture of a broken compensation, and all too often the cases are actually mistaken for a primary heart trouble, such as chronic myocardial disease.

Now, the great point to be learned in regard to these cases is this: Patients with contracted kidney almost never show any edema *until the heart breaks down*, and then the edema they show is a *cardiac edema*. Physicians are constantly at fault in this point. They feel that a kidney condition ought to have a kidney dropsy, but, as a matter of fact, a contracted kidney, because it is constantly excreting a greater amount of urine than normal, does not get a dropsy at all until the heart breaks down, and then it is a cardiac dropsy. So you are not justified in thinking that because this man has a nephritis his dropsy is nephritic—it is a pure cardiac dropsy, the cardiac insufficiency being, of course, the direct outcome of the high blood-pressure.

Management.—I want to speak to you a little about how you should handle cases like these two. How will you handle a case of contracted kidney with a broken-down heart? When you are called upon to treat a case of contracted kidney with a broken-down heart the thing to do is to forget the kidney for the moment, and treat it exactly as if it were a heart case, and put it on digitalis or, better still, the intravenous administration of strophanthin. I consider that the ideal treatment of a case of this kind. I think the Boehringer strophanthin is the most powerful of all the digitalis bodies, and regard it as most useful in this condition. Since it is exceedingly likely to produce necrosis of the tissues if you give it subcutaneously, I advise you to always inject it intravenously. This is a perfectly simple procedure—suck up the dose you want to give, bandage the arm until a vein stands out prominently, puncture the vein, and then inject the strophanthin very slowly, being careful that you do not get any in the surrounding tissues. The amount of Boehringer strophanthin that should be given should never exceed

1 c.c. in twenty-four hours. Personally, I almost never give that dose. I give half of it or, perhaps, two-thirds of it, because it is a drug that is very likely to cause trouble from overdose.

Now what will happen to the patient if he improves? This is a point again that is commonly misunderstood. If he improves his blood-pressure will probably go up. Isn't it a dangerous thing to push an already high blood-pressure higher? It is, but a patient with a contracted kidney is able to live only because his blood-pressure is high enough to push the urine through his kidney. Consequently, if he did not have a high blood-pressure he would not live. We see a man in between attacks of decompensation with high blood-pressure, and just as soon as he gets into an attack of decompensation down comes the blood-pressure. This man came in with a blood-pressure above normal, and you might think you must lower it; not at all. You must get him back into compensation, and the thing to do is to get the blood-pressure back where it was before decompensation set in. The thing to do is to treat him just exactly as if he had an idiopathic decompensation of the heart. That is, you must give him digitalis or strophanthin, as the case may be, until he is back in compensation. Just as long as the patient's heart is compensated, and he is going around with his heart acting normally, just so long does he have a polyuria and a small amount of albumin. Just as soon as the general blood-pressure and with it the pressure in the renal arteries goes down, and that in the renal veins rises, the amount of albumin goes up, and we have the typical picture of decompensation.

Now, gentlemen, when a patient comes into the hospital with a kidney lesion and a very high blood-pressure it is almost invariably simply a contracted kidney, and on top of the kidney a broken-down heart, but this increased quantity of albumin is apt to make one think that he has a case of acute nephritis. One commences to purge him and do everything except the right thing, which is to treat the case as a heart case until it gets back into compensation. The thing that keeps a patient alive with a contracted kidney is the high blood-pressure. So we can speak

of the hypertrophy of the heart and the raised blood-pressure as compensatory factors. Now, when that compensation commences to go, when his heart no longer is able to keep up, then we have the beginning of the end. When a patient with a contracted kidney has a cardiac breakdown it means the beginning of the end; not necessarily at once, but it means that his compensation is failing.

By not letting him work hard, by preventing him from running about and up and down stairs, we can sometimes keep him alive for a long time; but invariably when a heart gives out in chronic nephritis the end is not far off—possibly a year. Two years is about as long as we can keep the patient going when a broken-down heart has once developed.

I showed a case yesterday with a clean-cut history of a man who had evident earmarks of contracted kidney for at least twelve years back and was still in pretty good condition, but he has no signs of decompensation even now.

Summed up, it amounts to about this: The first case, a secondary contracted kidney, came on in a diseased kidney, came on rather rapidly in this case, following enormous quantities of drink. The second type, the primary contracted kidney, has taken many, many years to develop; he does not know he has a contracted kidney. This case is before us. The striking thing is that the kidney undergoes contraction, and as it undergoes contraction the blood-pressure rises, and as the blood-pressure rises the left ventricle becomes hypertrophied. So all these cases are associated with a high blood-pressure and hypertrophied ventricle. The functional value of the kidney gets less and less, as indicated in the test we have just made for you. As a rule, patients do not come into a general hospital until the compensation fails. The patient develops a cough, dyspnea, a swollen and tender liver, and edema of the feet. Above all, I beg you not to mistake the mechanical dropsy for a nephritic dropsy. It is dropsy that comes of the cardiac condition. Instead of having a polyuria, these patients have a scant urine, and, as the quantity of urine comes down, the albumin percentage goes up, so instead of having a large amount of urine

and a trace of albumin, we find a small amount of urine and considerable albumin.

When a patient comes in with high blood-pressure and broken-down heart you may be sure that it is an old case of nephritis and not an acute attack, and that makes a great deal of difference in the prognosis and treatment. The prognosis in the chronic case is bad, so far as life is concerned, but you can nearly always get them back into compensation in a first attack. Always when the heart begins to dilate the compensation gets worse; that is the beginning of the end. Don't make the mistake of looking at this case as acute nephritis. One well-known colleague has made the remark that nearly all the diagnoses of acute nephritis in the wards of a general hospital are wrong. The cases are very uncommon as compared with the number of cases of chronic nephritis. We have many, many cases of chronic nephritis, but I don't know of an acute case in the ward at present.

CASE OF HEPATIC ABSCESS. OBSCURITY AND DIFFICULTY OF INTERPRETING THE FINDINGS

I HAVE another type of abdominal case, a much rarer type, and one that is a good deal more difficult of diagnosis. It is one that puzzled us until last evening, when we finally managed to make out the diagnosis. The patient has been a puzzle to us for two or three weeks. He came in complaining of pain in the small of the back, and said that he had had it for a considerable period of time. The pain had not been very characteristic. It was a dull aching sensation of pain all around in the lumbar muscles. He came in on the 20th of February, nineteen days ago, complaining of pain in the right side of the back (I may say that this pain was not always localized on the right side, was sometimes on the left and at times even more severe on the left), chill, pain on urination, dark urine, and some loss of weight. Now he says that the illness began about two weeks before he came into the hospital, starting with pain in the right side—that it came on

every two or three days. For a few weeks before entering the hospital it had been worse, occurring every day. Previous to that time it would disappear for a couple of days at a time. The pain does not radiate to the groin or scrotum; has never been very sharp. He has been nauseated, but never vomited. Has never had diarrhea. The face, feet, and genitalia have never been swollen; he urinates rather frequently and has slight pain with urination; the urine is dark, but never any blood. Thinks he has lost about 10 pounds in the last six months. Has had no headache; says his feet feel a little cold, but that he has never had a chill. When he came in he had a little temperature—100° F.—which would rise to 101° F. at night, the pulse being about 100; the next day it was about normal; the next, 99.6° F., and it ran along for a week or ten days, going up to 99° or 99.6° F. in the evening, and the first of this month it celebrated by going to 101.4° F., then stayed at 100½° to 101.4° F. in the evening, with an occasional day off. A rather irregular temperature; never very high, never going above 101.6° F. The physical examination was absolutely negative in every respect, except that he seemed just a little tender over the back, not very much, but a little stiff in the left side, and a little tender over the lumbar muscles on both sides. Examination of the urine has been made every day since he has been in, and it was about the same for the first week, from 1018 to 1020, always acid in reaction, a trace of albumin, a microscopic amount of blood, and some hyaline and granular casts; and then on the 28th of the month we suspected that he might have stone. We had a ureteral catheterization done and he had a little blood for the next day; this then disappeared entirely. This we attributed to trauma in the catheterization. The amount of albumin is always a mere trace, perhaps $\frac{1}{20}$ of 1 per cent., next day a trace. The phenolsulphonephthalein test was made; the phthalein appeared first in thirty minutes, with pretty fair excretion as a whole. That did not throw much light on the question. The blood-pressure was a little below normal—110 systolic. The leukocyte count was 16,000 on the 22d of the month. Then he complained a little of some dyspeptic trouble; we thought because of the blood that

there might be a kidney stone, and had an *x*-ray made, but found nothing either by *x*-ray or fluoroscope.

We then gave him some test-meals, and found that the tests showed a good deal of hydrochloric acid, and that's about all. No amebæ or blood in stools or anything else worthy of note, Physical examination remained negative except as already noted, and we could not find a thing anywhere. Patient had a little temperature, and occasionally a little bit of albumin in the urine, an occasional blood-cell in the urine, and that's all. We suspected everything there was to suspect, but were unable to make any sort of a diagnosis. The next blood examination showed a drop in the leukocytes, and we made up our minds that there was probably a slight kidney infection, but there didn't seem to be enough in the urine to explain the general condition.

When I went over the back carefully a few days ago I thought I noticed a little bit of dulness down at about the eleventh rib and a little more tenderness over the kidney. There seemed to be a difference between the two sides, but this was not very well marked, and three days ago it was even less marked than now. It seems to me now that on the right side there is dulness somewhat higher than on the left. The right lung boundary moves with inspiration, but less freely than the left.

Now the Litten: He has a well marked Litten sign, not as well marked on the right as on the left, but still present. Let us ask ourselves what the possibilities could be. A man comes in with an obscure temperature, running up and down, with a history of having had it before, with a leukocyte count running from 16,000 to 24,000, and today 19,000, with an irregular temperature, as in a septic condition. Tenderness over the lumbar muscles on both sides, a little more marked on the right. Now what could we have to correspond with that clinical picture? A focus of pus somewhere. Where could it be? It could be in relation to the diaphragm—just above or below it; one might think of an empyema, but the man has had nothing to cause an empyema. Everything he has points rather to the abdomen. Difficulty in urination, dark urine, pain on urination, the negative chest findings, tenderness over the lumbar muscles—every

symptom the man has points to something below the diaphragm. The *x*-ray shows the kidney and subphrenic regions perfectly negative. On going over the area again last evening I made up my mind that there was something in his liver or in the subphrenic region, so I suggested that we put in a good long needle in the eleventh interspace behind, and we were rewarded by finding a lot of pus. The smears showed very little, and it looks like an old sterile abscess. Is it above or below the diaphragm? If above the diaphragm, it would be probably an old empyema, and the man should have had shortness of breath, pain on taking a deep breath, should have a history of pneumonia or something similar preceding. Then if it were empyema there would have been other symptoms, and it seems more likely that it is below the diaphragm, and that was our reason for going in where we did. There are only two places where it could be; in going in with the needle where we did there can be no reasonable doubt that we have either a hepatic or subphrenic abscess.

The important thing to bear in mind is that an abscess such as this may last for a long time and show very few symptoms. There may be simply a little pain in the back, and we often find that an abscess in this locality may go along for months and months and show almost no symptoms. I saw a patient not long ago in private practice with a subphrenic abscess where almost 2 quarts of pus were taken out, but there had been almost no symptoms. These perinephritic abscesses may last a long time and produce very few symptoms, and the intrahepatic abscesses may remain latent for an indefinite period of time.

Now I want to go over this patient in the axillary line with you and see what we can find, because I didn't have a chance to go over him very carefully this afternoon before we came down. Even now the resonance comes down to the eighth rib. On deep inspiration you can hear the change in note. That is almost conclusive evidence that we have to do with a condition below the diaphragm. The results of puncture are often uncertain; indeed, a needle puncture is one of the very uncertain methods of diagnosis. Almost any one can relate to you how he

has gone into a pus cavity time and again and found no pus; but, on the contrary, if you find the pus by your needle that settles it, and if you don't find it, that doesn't justify the assumption that it is not there.

In this case the etiology is obscure; it is probably not tuberculous. There are many cases where the pus has been there so long it has become sterile; at any rate, in the smears we found no organisms at all and no amebæ. We have, then, an abscess below the diaphragm, but whether in the liver itself or subphrenic cannot be stated with certainty.

The **treatment** is surgical; the pus must be evacuated. It is interesting to note two things: In the first place, at least two examinations of the kidney region by means of the fluoroscope and *x*-ray were made and all failed to reveal this abscess; the *x*-ray report sent down was that the diaphragmatic region was entirely negative. So we are not justified in depending on the *x*-ray in such cases. I think we should have made the puncture three or four days ago, and did suggest it, but the man's leukocyte count came down so suddenly that I decided to wait a few days more. I think the pus has been there for many, many weeks and possibly months. We shall send the case to the surgical service tomorrow.

GOUT. CARDINAL SYMPTOMS OF ACUTE FORM. ATYPICAL NATURE OF THE CHRONIC FORM

I HAVE here a case that is the type of case I like to show because it is one of the cases that goes begging for a diagnosis. The man is single, a laborer, Bohemian, forty-two years old; enters the hospital with a diagnosis of acute articular rheumatism. Comes in at the present time because of pain in the right foot down in the lower part of the great toe; a sharp, sticking, needle-like, continuous pain. Pain comes on and lasts for quite a while; sometimes comes on at night. He goes to bed and wakes up in the night with severe pain in the right foot. He has

had similar attacks for many years, recurring twice a year or three times in two years. He had been sick for about ten days, but is getting better now.

Here we have a man who has been having attacks of intense pain in the foot, particularly around the great toe, first on one side, then on both. He has been having them about twice a year to start with, and then every eight or nine months, and, as I said, it has been diagnosed as rheumatism. When he first came in the appearance was much more marked. You see the toes are red and swollen, although not nearly as much so as when he first came in. At first when I moved the joint there was crepitus, but not much; the soft structures are even now half again as thick as they should be. We find the foot sweating profusely, which is quite typical of this condition. When my attention was first called to him the area was as red as fire; the tarsometatarsal joints are still tender, but better than they were.

He has had it in the little finger of the left hand, and you see the joint here between the first and second phalanges is swollen and very tender. What is it? It is a perfectly typical case of gout. I don't know where you would find a more typical case. A man, a heavy drinker, comes along with a history of pain in the right foot, a greatly swollen, tender great toe; the attack comes on in the middle of the night with exquisite pain. There is not very much temperature—99.4°, 99.6° F.—very little temperature, then the trouble spreads up to the other joints of the foot, and then, after many years—he has had this for twelve years before—spreads up to the joints of the hand, attacking the little finger first.

As I say, these cases generally go begging for diagnosis. If one would look over the cases of "rheumatism" he would find many cases of gout. I think it would be found almost as often here as it is in England, and that is the home of gout par excellence. Gout is not a very rare disease; nor is it a very common disease, but it often goes begging for a diagnosis. If a man goes over his cases carefully with gout in mind, he discovers that he has a great many more cases than he had supposed.

Gout, as you know, is a disease of metabolism; it has nothing

to do with rheumatism. It is a disease of metabolism associated with the purin bodies. Gout is due to a perverted metabolism of the purin bodies, and the fundamental trouble probably lies in the failure to eliminate. That is, the history of the gouty

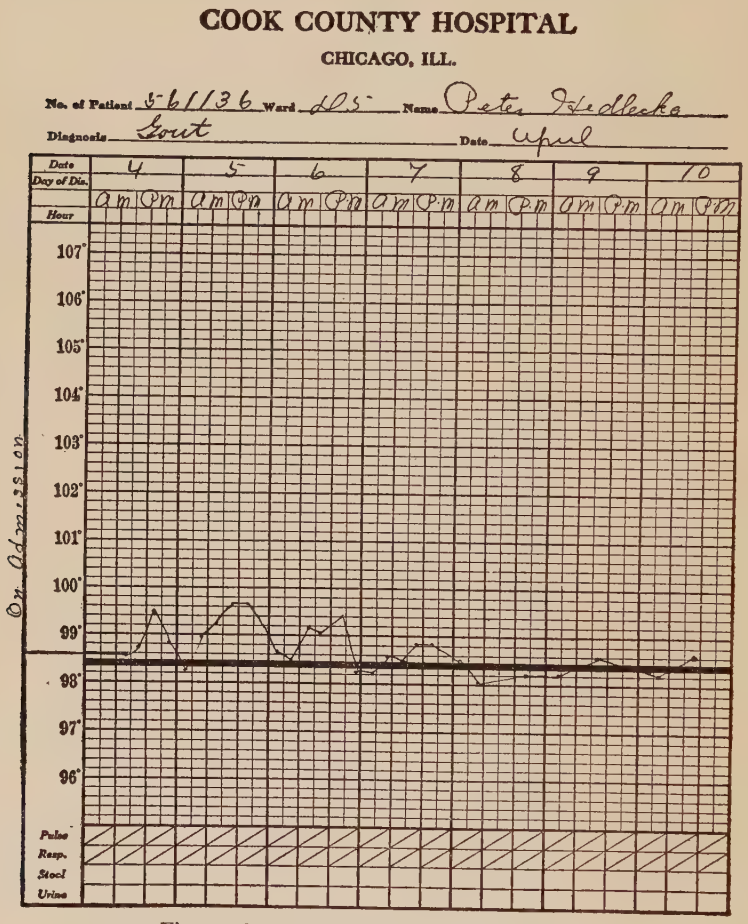

Fig. 5.—Recurrent gout, subacute attack.

process is that the patient is not able to eliminate sufficient quantities of the purin bodies, and we have a retention of the uric acid, and this retention gives rise to all the trouble. This is a perfectly typical case of subacute gout superimposed on an old

chronic case. When the first attack came on the patient perhaps had never been sick before. He wakes up in the night with a sharp, violent, shooting pain in the great toe. The temperature goes up a little bit, possibly to 101° or 102° F., and the pain is simply excruciating. In England this disease has become so common that it has become a typical English cartoon. In the morning the pain lets up after lasting for six, eight, or ten hours; the patient goes to bed the next night and has the same experience. Occasionally it disappears with the first night, or it may recur for a week and more. Now this attack goes away and the patient may not get another for many years, or may have another in six months. I remember one patient I had whose wife could always tell when he was about to get an attack of gout. He just got cross, felt mean, and she could predict almost to a certainty that he was going to have one of his attacks. Following these attacks the patient generally feels unusually well and unusually happy—probably at getting rid of this intense pain.

Now, the disease in time becomes chronic. The attacks get more and more numerous, and the condition spreads to other joints, and as the attacks get closer together they lose their typical character. The patient fails to get the typical temperature of the acute attacks, and after a while it goes into the definitely chronic stage, and the patient has it more or less all the time. Then it attacks the other joints —the distal joints —almost never the proximal joints; we rarely see it in the hip-joint. The next joints to be attacked in this patient were the small joints in the metatarsus. A favorite place is in the tendo Achilles; so we have it along the tendon sheaths and along the great tendons. It sometimes goes up into the knees and we sometimes get an exudate. The first point I want you to bear in mind is this, the acute attack of gout you can't mistake, even if you see it for the first time, provided you really are thinking about what you are doing. The chronic cases can be very easily overlooked, especially when you get it in the polyarticular stage. Here we have a man with the great toe involved, up here a joint or two on the hand, but no temperature at all. When gout has gotten to the polyarticular

stage it ceases to be a very typical disease, so far as acute symptoms are concerned, and may be readily mistaken for some other form of joint disease. On the other hand, when you find a rheumatism with five or six joints involved you find considerable temperature. When I looked at the patient's temperature chart and I saw there was little temperature, with so much joint involvement, it seemed very probable that we had to do with a gout, and that is what it has proved to be.

What is the pathology of gout? The pathologic deposits in gout consist of sodium biurate—first, in the cartilages; second, on

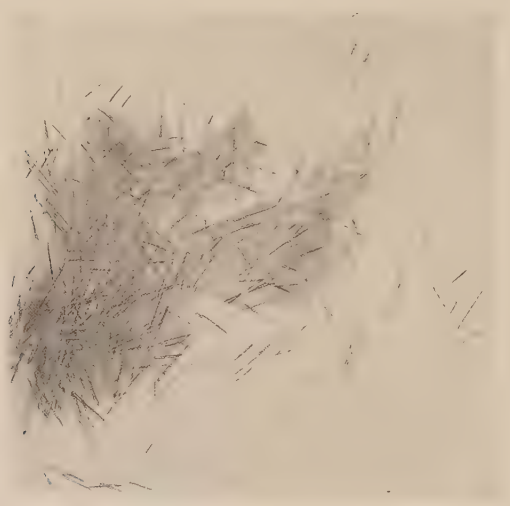

Fig. 6.—Crystals of biurate of sodium from gouty tophus.

the joint surfaces, and third, in the ends of the bones themselves and in the epiphyses. This can often be demonstrated by means of the *x*-ray. Brugsch considers that the third great diagnostic feature of gout: First, the gouty attack; second, the tophus; and, third, the Röntgen ray picture. *x*-Ray plates show absorption of the bone, which, of course, is most helpful. In the last few years, since we have had the *x*-ray examination, we have been able to demonstrate the changes in the epiphyses and in the bones. Now, these sodium urate crystals may be deposited in the cartilages, in the synovial membranes,

deep in the tendon sheaths, at the insertion of the tendons, and around the cartilages elsewhere, particularly in the ear. In this particular patient I have not been able to locate any in the ear, but you often find little white granules about as big as a pin-head or larger. These sodium biurate crystals are very fine and very numerous, and they are embodied in a chalky mass a little like cream, and are very characteristic. If we were to cut down on this patient's joints and take out one of those tophi, you would find just what I am going to show you here under the microscope. These joints practically never suppurate, although very angry looking, and it has happened a great many times that a surgeon has opened a gouty joint and found nothing. Occasionally tophi will develop and produce pressure necrosis and suppurate then. We have opened them and they do not show pus, but a definite, white, chalky mass with sodium biurate crystals, and this is the most certain diagnostic symptom we have in gout. They invariably mean that you are dealing with a case of gout. They are perfectly typical and perfectly characteristic.

A few words about the chronic signs of gout. As gout becomes more common it takes on many forms. In England you see many peculiar things—many, many things which are called gout which probably have nothing to do with gout. My advice to you in the diagnosis of gout is to hold fast to the cardinal points. First, either you must have it in the toe alone, or else starting in the toe and going to other joints later; about 75 per cent. of the cases start in the toe. A sudden onset is characteristic, usually starting in the night. Second, the appearance of tophi. Third, the Röntgen changes in the epiphyses—that is a point which I think is not sufficiently appreciated in this country. Lastly, don't make the mistake of calling a case of gout rheumatism when it becomes polyarticular. Here you have a nice type—a mono-articular case originally, which has spread to become polyarticular. I will show you the sodium biurate crystals here under the microscope.

FURTHER DISCUSSION OF CASES SHOWN AT PREVIOUS CLINIC

ABSCESS OF LIVER

I WANT to talk to you for a moment about a couple of cases which I showed you the last time. You will remember the one in which we diagnosed an abscess somewhere below the diaphragm. At my suggestion, Dr. Greer had gone in at the eleventh intercostal space, about 4 inches from the midline, and obtained pus; he used a large spinal puncture needle, which is the best thing to go in with. We sent the patient up for operation, and he was operated at 5 o'clock the next evening. I went over the case again very carefully after leaving here, but could find no reason for changing my opinion. The abscess was certainly below the diaphragm, but its origin we could not determine. We had a clean-cut Litten sign. Dr. Speed went in at about the site of the needle puncture; got in, but found nothing. He then went up a little higher and opened the pleura and found nothing; went down further, but still found nothing. You could see very plainly on the outside of the liver that there were changes there, but aspiration revealed nothing. He aspirated the liver two or three times, but got nothing; so then he put the question up to me as to whether he should take a pair of forceps and go into the liver, and I told him I was perfectly confident the pus was there; so he went in further and found 1½ quarts of moderately thick yellowish pus, practically sterile. In other words, it was an old abscess. The fact that it was sterile suggested, of course, the presence of the amebæ of dysentery, but we could not elicit any history of dysentery or anything that would throw light upon it, nor could amebæ be found in the débris.

The lesson to be learned is this: In the first place, an abscess may lie nearly latent for a very long time. The patient had been in the hospital before on account of it. He had been free from fever so far as he knew for a long time, and then this abscess would light up again; then he would go along with very little trouble except a slight pain in the back. I do not feel that

I have to apologize for the time it took us to make the diagnosis, because the man came in with the vaguest symptoms and history, and if the doctor had not been skilful enough to get the pus I should be hesitating about the diagnosis now. There was fully 1½ quarts of pus right up under the diaphragm when he put the forceps in. Remember the difficulty of getting anything with a needle. It was only after the doctor put the forceps in that he got the pus. The man is in fair shape now, but is too sick to bring down to the clinic today; he has a fair chance of pulling through, for it has been six days since he was operated.

CONTRACTED KIDNEY

Here is another case that I showed you last week—one of the two contracted kidneys. They were both in broken compensation, and I went into the question of compensation of the heart in kidney cases with you at some length. I said that the primarily contracted kidney never had any edema until the heart breaks down. A man can't have a polyuria and have an edema in the long run. A man can't have his pie and eat it—he can't have a polyuria and keep his fluids in the form of edema. I emphasized, and want to emphasize again, that the dropsy in these two cases was a mechanical dropsy. You remember that I showed you the two patients with their feet enormously swollen, their backs swollen, etc. I told you that the proper thing to do was to put them to bed and ignore for the time being that they had a contracted kidney. Treat them as you would a case of heart disease—put them to bed and inject digitalis intravenously.

Now I want to show you the result of that therapy; that is all this patient had done to him. You can pick his skin up very nicely; the skin over the abdominal wall is very soft, no signs of any edema; the back was markedly edematous, but that has all disappeared. That is what you expect in a case of decompensation with contracted kidney if the patient has enough "kick" left in the heart to respond to the treatment. The blood-pressure goes up—let us see, the blood-pressure was 160 systolic and 145 diastolic. You would expect the systolic to go up and possibly the diastolic to go down. The pulse-pressure, that is, the dis-

tance between the systolic and the diastolic pressures, might be called the useful net blood-pressure, and the increase of this is an important factor in the restoration of compensation. Remember I emphasized, and want to emphasize again—don't worry about the blood-pressure going up; it must go up if compensation is to be restored, except in the cases of high-pressure stasis.

Now we have the patient back in compensation, practically in what is *for him* a normal condition. Now, we must not forget that whenever we have a contracted kidney with a heart that is broken down in its compensation the patient is in the last stage. You may be very sure that it marks the beginning of the end, even if this be somewhat protracted. Where is the danger in diagnosis in a case like this? The danger is that you overlook entirely the renal component. The high blood-pressure has, of course, produced the cardiac dilatation, and made the case look like a pure heart case. This patient has been treated exclusively as a heart case and this is the result. Now, don't forget it—these cases are messed up badly ordinarily. Put them to bed and do not purge them and sweat them, but treat them as cases of broken-down heart, and get them back in compensation. We injected digitalis and strophanthin intravenously in pretty large doses, and he wanted to go home three or four days ago because he felt so much better.

Now, you remember, I showed you a second case as near like him as two peas in a pod—his edema has disappeared entirely. You remember that he was a pretty hard drinker, and as he is now showing signs of delirium tremens I did not feel quite justified in bringing him down. His edema has entirely disappeared; his blood-pressure went up a little, but so far as his compensation is concerned he is all right. He still has his nephritis, of course; but his compensation is all right, and he is as greatly improved as is possible in his case.

Now, one last point. I urge upon you to use digitalis intravenously on a case like this, or strophanthin in the same way. When you give it intravenously you know that it gets directly into the circulation; you know that it is not floating around some-

where in the gastro-intestinal tract. When he has edema of the stomach and intestinal walls it is doubtful if any very considerable amount will be absorbed. Intramuscularly is better than oral administration, but if you give it intravenously you know that it is effective, and it represents the difference between efficiency and inefficiency in treatment. I prefer the digipuratum myself, or the strophanthin of Boehringer (intravenously).

This type of patient that just went out is likely to do this: He will probably go out of the hospital, and in two or three weeks or a month he will come back again. He has not much intelligence, and, in spite of good advice, he will go out and work hard and drink a lot and throw all sorts of strain upon his heart, and in three or four weeks he will be back. In private practice, where a patient is under careful observation, that kind of a case can live for a good many years. Some of them can stop all their work and take the strain off their hearts, and they will live for three, four, or five years; but in cases like this a year or two is about the length of time that we can expect them to live. They get to be old friends of ours, come back to the same ward, and are sent to the same man they were under before; so we have abundant opportunity to observe them.

CHRONIC GOUT WITH EXTENSIVE JOINT CHANGES. DISCUSSION OF THE METABOLISM IN GOUT.

Now, you will remember the last time I showed you a case of gout which interested me very greatly, and, as you know, while gout is not so very rare, it is not so very common. A hospital like this will possibly have a dozen or fifteen cases that will be recognized in a year; so when I went up and found that we had acquired another case just like the other one I was somewhat surprised. Let me show you the old case for a moment and show you what he has done in the meantime. You remember the patient's condition—you see that he has an acute gouty attack here right now, the skin is reddened and the joint very

Let us take up the differential points of gout as a whole. In the first place, the onset in at least half the cases is in the great toe, with or without some other joint being involved. The great toe by itself is attacked in fully half the cases. Second,

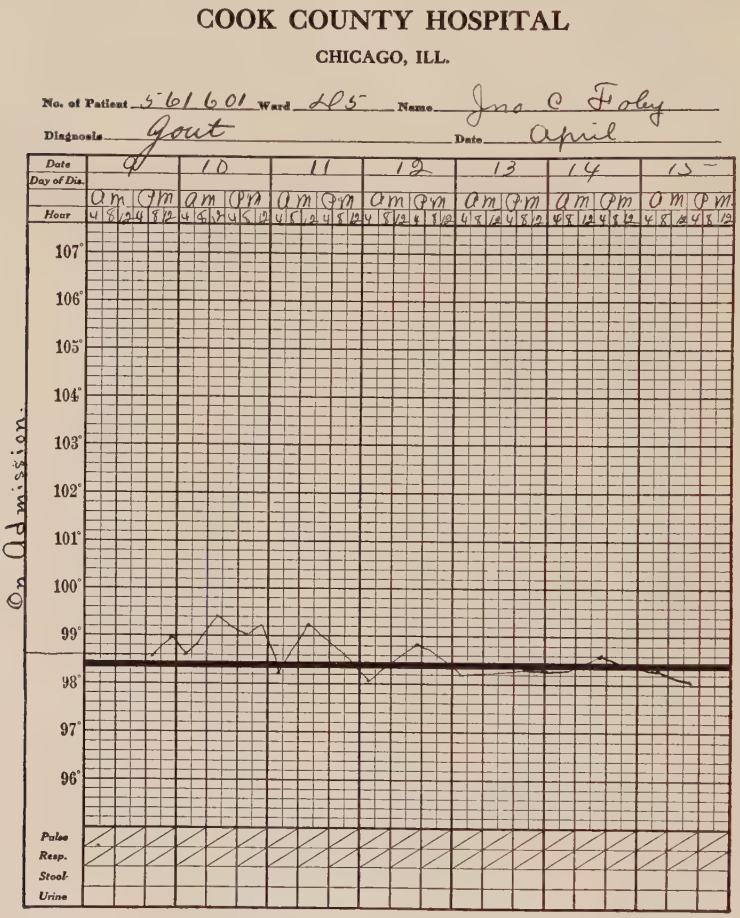

Fig. 9.—Temperature chart of gout case.

the onset is generally at night. Third, there is very severe pain. Fourth, the typical attack is febrile to start with; that is to say, when the first attack comes on there is generally temperature with it; the temperature may reach 102° or 103° F.,

subsiding, let us say, in ten or twelve hours. Next, the patient feels, as a rule, extremely well after an attack. Ordinarily the patient has a recurrence the following night or on several successive nights.

DR. WILLIAMSON (to Patient): Did you ever have a recurrence like that?

PATIENT: Yes, I had one on Saturday, and then it went away, but it came back on Sunday and I could not walk at all.

DR. WILLIAMSON: Exactly; that is perfectly typical; comes on and goes away, and the next night it comes back. Now, after a man has had six, or eight, or ten, or twelve attacks, it has gotten past the acute stage to the chronic, and there are many changes. In the chronic cases the onset may be anywhere, but particularly in the distal joints and the small bones of the metatarsus. It very seldom appears in the hip or shoulder. As the cases get more chronic the onset may come on at any time and becomes less typical—*i. e.*, comes on more slowly and disappears more slowly. The pain is not so excruciating in the chronic cases and such cases become afebrile. I want you to notice that in both these patients there are a number of joints involved. The first patient had several joints in the feet, and this patient has several in the feet and the hands as well, *but still neither of them has had a temperature. So the combination of great pain and little or no temperature is characteristic of gout.* Gout may have temperature, but more often than in any other disease there is a lot of pain and still no temperature. Now, if I brought in a case of real acute articular rheumatism with even one joint much involved the patient would probably have a temperature of 102° or 103° F. So as between rheumatism and gout the absence of temperature along with involvement of several joints invariably speaks in favor of gout. That is a point you will do well to hold fast in mind. These are the differences between the acute and the chronic attacks of gout.

Now let us compare one of these cases of gout with one of acute rheumatism. I want to show you a case of typical polyarthritis rheumatica along with this gout. Here we have a patient with typical rheumatism, and we find almost the same

sort of condition; there is just about the same amount of tenderness and just about the same amount of involvement. But here we find a very different sort of a story: we find this patient's temperature reaches 102.6° F.; he has just one joint involved and yet he has a temperature of 102.6° F. This is an acute attack, the first one he has had. The history is that it started on March 14th, when he was taken sick with a chill and sinking sensation with a pain in the right hip, which then extended down the leg and settled in the right ankle. Swelling occurred simultaneously, and the ankle became swollen and red. I only want to contrast the temperature in this case with the absence of temperature in the gout.

Now I want to say a few words about the *x*-ray changes in this case of gout. That is one of the new things that has not broken into the books very extensively. I recommend you to read Brugsch's article on this. He has studied a large number of cases, and comes to the conclusion that it is only with the *x*-ray that we can appreciate the changes. What are the changes that occur? First, there is a precipitation of the insoluble sodium biurate in the cartilages, in the superficial layers of the cartilage, and this occurs not only in the cartilages, but in the bones themselves. Now, where the biurate of soda is deposited in the bone itself, it causes absorption of the bony tissue, and whenever that occurs it lets the *x*-ray penetrate a little better, and this can be shown in a well-marked case with the *x*-ray. I have had very attractive pictures taken of both these cases and will show them to you.

There are three or four things in gout which are typical, and I want to take these up with you one by one. In the first place, the acute attack. When you can get a clean-cut history such as we have here it is typical of gout. Second, the tophi. Third, the *x*-ray changes, when they are present; and, fourth—most typical, most characteristic of all perhaps—are the changes in the uric-acid metabolism.

We have already discussed the acute attack. We have already seen that these cases can be diagnosed on the acute attack alone. Second, the tophi—what are tophi? They are

Fig. 10.—Tophi in ear (gout). Case of Dr. Charles Spencer Williamson. Drawn from life.

deposits in the ear, in the joints—particularly of the ankles. These deposits are made up of sodium biurate; they are hard, whitish masses with a chalk-like consistency, and frequently in the beginning are rather soft. Tophi occur in only about half the cases—so you must not think that a tophus is essential to gout. It is not essential: if it occurs it is a conclusive sign of gout; if it does not occur, all you can say is that the case has not the tophus form. I showed you what the tophi were made up of the last time; those long, needle-like crystals. In this ear we found none. In this one we found a deep-seated, hard nodule, just as hard as it could be; so we took a bistoury and pried loose a little nodule which looked for all the world like a tophus, but it was not. I want to show you this because it is one of the common mistakes that can be made. I will show it to you under the microscope, and you will see that it is a little fibroma, and you would have made a mistake in diagnosis, and a very bad mistake, to have taken that for a tophus. I was sure it was not a tophus before we got it out because of the great hardness. So don't jump to the conclusion that every little lump you find in a man's ear is a tophus. They are very likely to be fibromas. There is that interesting feature about this case, namely, that he had this little fibroid which looked like a tophus, but was not one.

Now the changes in the joints. We will put the two pictures from this patient here in the shadow box and you can see the deformities very well. You can see the peculiar mottled look here which shows the infiltration in the cartilage, and at the head of the bone very distinctly. It shows a great deal more change than you would think possible.

Now we will show you the plates from the other case. Here we see the changes, but not quite so advanced as in the last case. You can see the infiltration here. You can see the deposits here in the head of the metatarsal bone. You notice around this joint of the great toe the infiltration that looks like a little cyst. That is the infiltration of the sodium biurate crystals. Now I don't want to be quoted as saying that it is possible to pick up a plate and say, "That is a case of gout,"

and I think Brugsch goes a little too far in saying that they are typical of gout. In fact, I am very sure that no one without knowing anything about the patient could pick up one of those plates and say positively that it was gout, differentiating it from arthritis deformans, although they are quite characteristic.

The accompanying pictures (Figs. 11–14) are from a case of chronic gout with acute attack. They are probably the prettiest *x*-ray pictures of gout that you have ever seen. There, for example, are the changes in the metatarsal region of the great toe. You can see how the bone itself is degenerated, showing a very advanced degree of gouty change. In the other joints of the foot one does not see it plainly in this picture, so I will get another view of the same foot. Here you are looking at it on edge, so to speak. Look at the deposits in the bones. They are rough and infiltrated, and then as you look at the tarsal bones you will see how they are swollen and infiltrated. This is the other foot, which shows almost duplicate changes. In the distal end of the metatarsal bone of the great toe you can see how the process has infiltrated the bone, producing all this change. I don't see anything in the other joints in this particular picture. Here we have the lateral view of the other foot which shows the same changes, and here we have one of the prettiest pictures you can imagine, and that is the right hand. Look at the beautiful infiltration in the thumb—as pretty as anything you can think of; look at the phalanx—you can see as pretty and typical infiltration as one ever gets to see. There was one spot in one of these plates that showed a little bit of infiltration with sodium biurate, which is one of the most typical signs of gout and more characteristic than any of the other findings.

I brought down a case of arthritis deformans to show you the difference in cases of this sort. Arthritis deformans is a very different sort of a disease. It used to be called rheumatic gout. It has nothing to do with rheumatism and has nothing to do with gout. I know of no reason for calling it that. In the first place, the acute attacks hardly simulate gout at all. They do not come on particularly at night. Second, they don't attack the periarticular tissues as gout does. It is not often that you will

Fig. 11.—Gout.

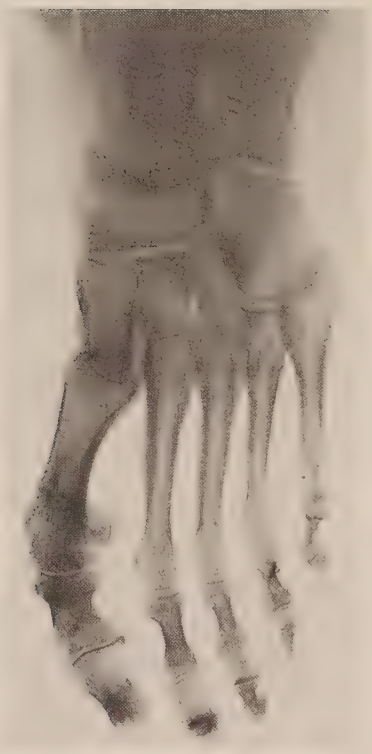

Fig. 12.—Gout.

Fig. 13.—Gout.

Fig. 14.—Gout.

Fig. 15.—Gout.

mistake an arthritis deformans for gout. This patient has a well-marked, chronic case of arthritis deformans—first, he has no temperature; second, there are no peri-articular joint changes to amount to anything. The ankle-joint is tender, extends down a little way perhaps on to the metatarsus, but there is no inflammation. It does not have that angry look that the other case had. You never find tophi in arthritis deformans. It is pretty tender here, but there isn't any temperature, no swelling, and no tophi. As you look at his right hand you see the lesions of a perfectly typical case of arthritis deformans. You don't find that swollen, red condition which we just saw in the two gout cases. The metacarpal and the interphalangeal joints are all involved. This always speaks against rheumatism and speaks ether for gout or arthritis deformans. We see some change in the elbow. It is a little thickened, and we see the atrophy in the forearm. The muscles are highly atrophic. It looks like a child's arm. So the combination of this old, chronic joint change with marked atrophy, little or no peripheral involvement, little or no temperature, speaks against gout and for arthritis deformans. If this were gout we would almost certainly see the great toe involved. I think I have never seen a gout with such extensive involvement as this case without the great toe being involved.

So you see many cases come up in which you may be in doubt. Bear these points in mind—in the first place, always ask particularly if the acute attack came on at night and let up the next morning. Remember that the patient will probably tell you that he stubbed his toe if he thinks it comes from something like that. Then, as the case gets older, look out for tophi. Then, after the case gets still further along, look for the *x*-ray changes. You need not expect to find these until there have been several attacks, for it takes some time for the sodium urates to change the joints or bones to a sufficient degree to be recognizable with the *x*-ray.

Now, there are two or three guiding principles in the discussion of these changes that I want you to bear in mind. There probably is no such thing as a real chronic rheumatism. I am not at all oblivious to the fact that there are probably many

cases diagnosed every day, but these cases are probably either arthritis deformans or the chronic infective arthritis that we find with local infections. I am assuming that you know how to look for the various foci of infection—in the tonsil, the teeth, the tubes in women, the antra, gall-bladder, appendix, etc.

Now I come to what I really want to be my topic for the day —*the study of metabolism in gout.* Let us just take a preliminary canter and discuss gout and what we know or believe about the essence of gout at the present time. I can't go into this in any detail, but just a little bit to sketch the subject for you. Gout has to do with the metabolism of the purin bodies and the cells. You will remember that we have, in the nuclei of the various organs of the body, substances which go under the general name of nucleoproteins, and with these nucleoproteins gout has to do. We find that these nucleoproteins are split up in the animal body, being acted upon by a group of ferments known as the nucleases. These break down the nucleinic acid into the two substances known as adenin and guanin. Now, under the influence of another group of ferments, known as the desamidases, these two substances are still further broken down into xanthin and hypoxanthin. Then we have a group of oxidizing ferments, and under the influence of these oxidases the hypoxanthin and xanthin are further oxidized into uric acid. Lastly, there is a fourth ferment (uricolytic in nature), known as uricase, which converts part of the uric acid, not all of it, into ammonia and urea. There is evidence to show that this ferment exists in man as well as in the lower animals.

The nucleoproteins are most marked where we have the most nuclei—in the thyroid, the thymus, the liver, the spleen, the kidney, the pancreas. These are the organs which are rich in cells. The consequence, as we shall see when we come to the subject of treatment, is that these are the things which we have to consider in the treatment of uric acid.

What do we know definitely about gout? We know, first, that it is associated with the uric-acid metabolism. Second, we know that the local changes are due to the deposits of sodium biurate in the joint. That's about all that we do know with

accuracy. Now let us see—uric acid can come from two sources: we speak, first, of exogenous uric acid; second, of endogenous uric acid. The exogenous part is brought into the body through the purin bodies. If we put the patient on a diet which contains none of those things—on a purin-free diet—he still secretes uric acid and we call this the endogenous. This is the uric acid which the patient manufactures from himself on a purin-free diet.

Now, wherein does the gouty patient differ from the normal individual? In regard to his endogenous uric acid. Suppose we put our gouty patient on a purin-free diet and now we study the uric acid. These two cases are on a purin-free diet; we shall keep them on it for three days, and then we will collect the urine and have it carefully examined quantitatively. In a normal individual we will never find any uric acid in the blood on a purin-free diet. The gouty patient, on the other hand, has a retention of uric acid in the blood, although it is sometimes a hard matter to find it. The gouty patient differs from the normal patient in the fact that he has uric acid in the blood on a purin-free diet. Now the urine: During the acute attack of gout two things will happen. There will be an excess of uric acid in the blood and a diminution of uric acid in the urine, and after the attack the amount of uric acid in the urine goes up. There is another thing which helps you to make a differential diagnosis. If we feed the patient substances which will make uric acid, what do we find? The normal patient excretes it rapidly, and the gouty patient very slowly.

There are three possibilities to consider: first, the gouty patient may manufacture too much uric acid; second, he may fail to convert it; third, he may fail to eliminate it. The trouble with the gouty patient is that he has some deep-seated fault in his metabolism by which he fails to eliminate the uric acid. That explains a great many things, doesn't it? It explains why on a purin-free diet the gouty patient has uric acid in his blood, and why the normal patient has not. It shows us why a gouty patient after an attack has uric acid in his urine in excess of a normal patient. It shows how the normal patient excretes it quickly, and how the gouty patient takes much longer.

From the practical standpoint, what constitutes a purin-free diet? In choosing a purin-free diet I will tell you practically how you go about it. A purin-free diet, as I like to use it, consists of milk, cream, cheese, bread, of course, eggs, butter—preferably not salted—such cereals as oatmeal, sago, rice; in fact, nearly all the cereals. These constitute our ordinary purin-free diet. Sugar, of course, is allowed, and such breadstuff as crackers. I will have to refer you to the tables to find what vegetables contain purin and which do not, but this is the purin-free diet which we ordinarily use.

Now so much for the metabolism of gout; a few words about the *treatment of gout.* During the acute attack there is not much to be done except the symptomatic treatment, unless it be a good dose of morphin if the pain is very severe. The joint should be elevated and the patient kept very quiet. I like to give a fair amount of codein and put on a bandage; that is about all that can be done. Colchicum has a great reputation; atophan is advised by the Germans, but I have a great deal of skepticism about its value. If our knowledge of gout is correct, and if it all centers around the purin bodies and the uric acid, and if the trouble with the patient is that he fails to convert or eliminate uric acid, it seems to me that the one clean-cut thing to do is to take away from the patient those substances that produce uric acid. The thing that should be absolutely forbidden is an excess of protein material of any kind. My own opinion is that it is best to eliminate meat entirely and give them the necessary proteins in vegetables. You must not starve the patients, but, on the other hand, you must be careful not to give them more than they need.

So, I should say that the salient points would be: first, avoidance of substances rich in nucleoproteins; second, substitution of vegetable for the animal proteins. Don't forget, when we forbid substances rich in nucleoproteins we include among them such things as beef-extract. These should all be cut out of the patient's diet. Third, restriction of the diet to the smallest amount necessary to keep the patient in metabolism. I think that is one of the keynotes of the whole thing—not only the

quality, but the quantity. Most of us, as a matter of fact, could keep our weight on a considerably smaller diet than we now have. The diet which the gouty patient should take should be the smallest amount which will keep him from losing weight—that is the proper thing for him. The next thing is the exercise, and a gouty patient should exercise pretty freely. When you see a patient having his first attack of gout you can do an immense amount to lessen the attacks. Sit down and have a heart-to-heart talk with your patient. Explain to him in as non-technical language as possible what he has to do and why he has to do it.

The next thing is the relationship of alcohol to gout; it is a doubtful one. We know that drink has a very marked effect in the production of gout; the probability is it comes from the general damage it does the individual, and the fact that the individual generally takes a much larger amount than he can consume. A patient was in my office yesterday who eats very little, but consumes a large amount of alcohol. He is a Southerner and whisky looks good to him. A man who takes four drinks a day, of one ounce each, does not consider himself a hard drinker. The average business man will go out and consume that amount every day; that means about 110 calories per drink. When he takes about four drinks a day he has taken almost 450 calories in drink. When he takes seven or eight drinks a day he gets 700 to 800 calories. In many cases you can do much good if you can get your patient to stop drinking. I think the great thing that has to do with gout is that we are taking in a large number of excess calories in drinking. I think it should be entirely eliminated. If a man does not want to get over a severe disease like gout badly enough to give up the drinking habit, he had better have the gout. The alcohol certainly does him no good, and he is much better off without it. If he will stop drinking he will get along very well. If you have your patient under good control you will find no difficulty in having him carry out this sort of a diet. Of course, they lapse from grace every now and then, but, on the whole, they get along very nicely.

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

INFANTILE TUBERCULOSIS. ORIGIN, DIAGNOSIS, AND MANAGEMENT OF CASES

THIS morning I want to show you a little girl six months old, a full-term baby.

History.—*Hoffman, Anna*, aged six months.

Admitted 2–28–15; discharged 3–9–15 (released).

Diagnosis.—Pulmonary tuberculosis.

Present Complaint.—Baby has not been well for last three or four weeks; has had a little fever and coryza and a little difficulty in breathing. Has been taking food regularly and bowels have been normal; occasionally a laxative has been given. Has been vomiting occasionally for the last two weeks, especialy after eating. For the last two days there has been increased difficulty in breathing and fever has been higher and the vomiting worse. Child has been restless and slept but little.

Past History.—Full-term baby, uneventful delivery. Breast-fed for first month, since this she has been fed the breast one day and the bottle the next.

Family History.—One other child, father and mother living and in good health. No history of tuberculosis.

On admission, temperature 104.2° F.; pulse, 124; respiration, 28.

X-ray examination shows areas of infiltration over both lungs.

2–28–15

Blood Examination.	Differential Count.
Hb. 80; R. B. C. 4,365,000; W. B. C. 36,200.	
N. 72	
SM. 16	
LM. 8	
T. 4	Cells counted, 100.

Von Pirquet—positive.

Parents signed a release and removed child from hospital.

This baby, as we heard, is six months of age. The points in its history are that it is breast-fed; the mother admits that she herself is thought to have some tuberculous affection. The baby was brought to us a week ago yesterday afternoon at the out-clinic, with a history of having had a high fever for some time; she was breathing rapidly and there was some dyspnea. We made a very thorough inquiry into her history, and found almost everything negative except for the points mentioned; we noticed that the child seemed exhausted out of all proportion to the symptoms. Since admission she has been running a moderate temperature—it was 104.2° F. shortly after admission, then fell to 101.6° F., and at midnight of the same day was 99° F. The fever chart represents the course of the temperature during the patient's entire stay at the hospital.

Now, on examining this baby we find that the face is pale, the head shows no craniotabes, the fontanel is unusually small, the sutures are nearly closed, though the baby is only six months old. The face is slightly edematous (puffy and swollen about the eyelids). There are no enlarged cervical or supraclavicular lymph-nodes, nor are there any nodes to be noted in the axillæ or groins; there are no enlarged superficial veins on the thorax. You notice as the baby breathes that there is considerable epigastric retraction, the breathing is rapid and involves a good many of the accessory muscles of respiration. Watch the muscles of the neck as well as the alæ nasi. The neck muscles are in rapid action. The alæ nasi are dilating frequently. I should like to call your attention to a very offensive odor about the child, which makes one think of a gangrenous condition of the lung.

On further examination of the chest, percussion of the anterior surface shows no deviation from the normal; percussion of the sternum over its superior portion and over the region of the thymus gland is negative. The heart borders are not enlarged. Posteriorly, one notices on percussion that there is an increase in the area of dulness between the scapulæ. On auscultation

Fig. 16.—Skiagram showing pulmonary infiltration on left side.

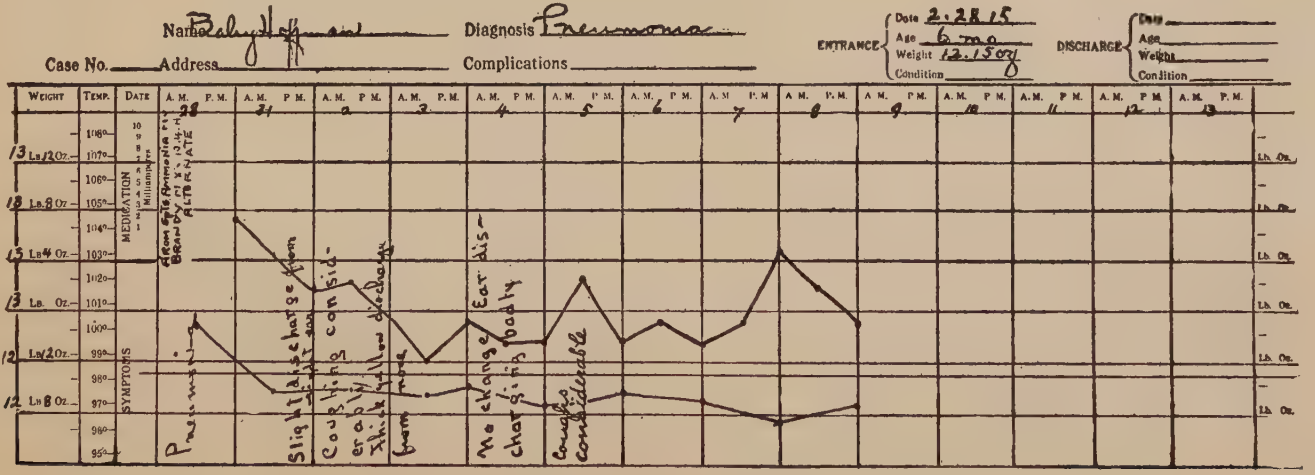

Fig. 17.—Showing daily variation in temperature curve. Tuberculosis of lung.

one hears a few râles over the dull areas, a normal respiratory tone, and a peculiar sharp, high-pitched respiratory note which suggests expiratory crowing. Sounds like those of a moderate expiratory cough are heard throughout the chest. There is no evidence of bronchial breathing. The heart tones are normal. We may say at once that the febrile course, the expiratory crowing, the dulness between the scapulæ assure the diagnosis of bronchial gland tuberculosis.

The abdomen is very slightly distended; the spleen is just barely palpable. There is nothing about the genitalia or extremities. The subcutaneous fat is tending to disappear and the baby shows a moderate degree of emaciation. There is no edema, no ascites; the skin retains its elasticity and apparently normal turgor.

On the right arm you will notice two areas that show a slight central depression and considerable redness—the result of the von Pirquet test, which was positive in both instances. One inoculation is probably a week old and the other is more recent. In the more recent one the upper inoculation appears like a large welt or hive, with a red areola probably $\frac{1}{8}$ inch in diameter. The lower one is somewhat smaller, but equally indurated; the central point, that is, the control, remains unaffected. Then, in addition to this, a week ago an intracutaneous test was made, the so-called "stick reaction," one injection of 1 : 50,000 of old tuberculin, one injection of 1 : 10,000 of old tuberculin, and one of normal salt were made. This reaction is made between the layers of the skin, and is a very delicate test. The intracutaneous reaction was positive and showed different degrees of intensity. The redness has nearly disappeared now, but even at the end of a week it shows some discoloration and induration. How many of the class have seen a case of infantile tuberculosis? I may say to you that this represents a type of disease that is not altogether uncommon in infantile practice. The cough is characteristic in a way; it is paroxysmal, and in a good many respects resembles whooping-cough, though the crow is expiratory in bronchial gland tuberculosis. The most interesting thing about the cough is the "expiratory crowing."

Tuberculosis is an interesting and important chapter in pediatrics or infantile medicine, and it occurs not infrequently. According to the statistics which have been made at various clinics, we are informed that tuberculosis during the first three months of life is uncommon. Von Pirquet found, after examination of 1000 cases with the von Pirquet test, that about 4 per cent. of children tested from the third to the sixth month were tuberculous, and that in the months and years following the disease occurred more frequently. He found, by the use of the von Pirquet test, that 70 per cent. of all the patients up to fourteen years of age who came to him showed a positive reaction. Now, that does not mean that all of these children who gave this reaction had an active malignant tuberculosis, but it means that in the bodies of these children there was some tuberculous focus, possibly latent, which might light up as an active process at a later time.

Now, possibly the most interesting point we have to ask ourselves is, If 4 per cent. of children under six months of age are tubercular, how do they become so? Is tuberculosis congenital? We have already spoken of the origin of tuberculosis, and we answer, Yes, a baby may be born with tuberculosis. Is it conceptional? Is it transmitted in the same way, for example, that we assume syphilis to be transmitted? Is it contained in the ovum or the sperm? It has long been held that an active tubercle bacillus is fatal to the germinal cell, and, therefore, we do not believe that tuberculosis is conceptional in transmission. But there are numerous cases reported from well-conducted clinics and laboratories where it has been shown that babies come into the world with tuberculosis; tuberculous lesions were plainly seen in the placenta. Or it has been shown in a few cases that the body of the baby swarmed with tubercle bacilli. But these cases are rare. Suffice it to say, that in order to establish the diagnosis of congenital tuberculosis it is necessary to find the tuberculous foci in the placenta or the organs and tissues of the infantile body. This has been done in some cases. If we refer to the statistics of von Pirquet and Hamburger, we learn that tuberculosis during the first few months of life is rare. How are

babies commonly infected with tuberculosis? Probably through another person. You know that the greatest menace so far as the dissemination of the tubercle bacillus is concerned is the "tuberculosis carrier," *i. e.*, the individual with an open tuberculosis is the greatest menace to the health of other individuals. The tuberculous mother who comes in contact with her offspring, and is constantly eliminating or giving off from her body tubercle bacilli, is very likely to infect her child; or the tuberculous nurse or any other member of the family may be a tuberculosis carrier and convey the disease to the infant. It is most frequently acquired from some person in the household. In what way does the tubercle bacillus enter the body? In a very large percentage of cases through the respiratory tract. Ghon, now Professor of Pathology at the German University of Prague, and Albrecht, of Vienna, have shown that the largest number of cases of tuberculosis in infants are bronchogenic; that is, the tubercle bacilli are inspired or inhaled through the respiratory organs and find their way into the parenchyma of the lung, and hence the parenchyma of the lung comes to contain the initial lesion of tuberculous infection. These authors are very insistent in their writings that this infection of the parenchyma of the lung may be a small indurated area which on autopsy may look like a section of the bronchus. The microscope shows an inflamed area containing infiltrating cells. This initial lesion resulting from the bronchogenic type of tuberculosis may very soon cause all adjacent bronchial and mediastinal glands to become enlarged, and we have the commonest clinical manifestations of infantile tuberculosis. Now that is one way in which the tubercle bacillus gains access to the infantile body—probably the commonest.

There is heated discussion in various parts of the world as to the mode and manner of infection with the tubercle bacillus. As a rule, the human body is infected with human tubercle bacilli; sometimes with bovine bacilli. We are told on authority that bone tuberculosis is frequent in Scotland, and that in Edinburgh intestinal tuberculosis is common. Specimens of cows' milk were examined by Mitchell. He found in the first instance that 10 per cent. of the samples examined contained tubercle bacilli,

and in a second series Mitchell showed that 20 per cent. of the samples contained tubercle bacilli. It was also shown by this investigation that 84 per cent. of children two years old and under had been fed unsterilized cows' milk since birth. It is not probable that the disease is acquired in a single way, and the best proof that the mode of infection must vary somewhat is the variation in manifestations in different localities. It must be true that in Austria and Germany, where milk is universally boiled, tuberculosis is largely bronchogenic in origin (bronchial gland tuberculosis), whereas in Scotland and England tuberculosis is very frequently intestinal in origin, resulting sometimes in bone tuberculosis. So it is possible that in the latter countries the disease is bovine in type, derived from unboiled milk; and in the former countries the disease is probably bronchogenic in type, derived from tuberculous human beings. So much, then, for the mode of infection.

I have already referred to the frequency of this dread disease, and the fact that tuberculosis occurs more commonly in crowded communities and in individuals that live under poor hygienic conditions. The statistics which I offered from the von Pirquet and Hamburger clinics show the frequency of tuberculosis among the poor (the poverty stricken), but they do not represent the conditions that prevail among all classes. In the large continental cities tuberculosis is very common among infants and children. Possibly the most frequent form of tuberculosis is the bronchial gland variety. If it is true, and it probably is, that the largest number of infections occur through the respiratory tract, then we would naturally expect the bronchial gland tuberculosis to be the most common variety. The period of incubation is difficult to ascertain. It may not be longer than a week. We know this with reference to infantile tuberculosis as we know it with reference to all infectious processes. The baby's resistance against infection is minimal—is not to be compared with that of an older child or an adult. We have already pointed out that any baby who falls ill with a serious infection, such as scarlet fever, for example, offers an unfavorable prognosis as compared with an older child. The same applies to erysip-

elas. A newborn baby with erysipelas presents an absolutely unfavorable prognosis, and an infant with diphtheria is likely to have the most severe complications and the most general symptoms of infection and toxemia.

So it is with tuberculosis. The little baby under two years of age does not tend, as a rule, to make this tuberculous infection a localized affair, but almost always it becomes a generalized infection. There is a marked tendency to rapid and general dissemination of tuberculous lesions throughout the body. We believe that when the tubercle bacilli have gained access to the bronchial glands the antibodies begin a process of activity that tends to digest or in some way or another absorb not only the toxins, but, more particularly, the organisms themselves. During this process of digestion of the organisms by the antibodies, toxic products are set free, and we believe that at this point, when the toxic products are liberated, the generalized infection begins. These antibodies are very soon exhausted, and if the bacilli be present in the very center of the gland they are, as it were, inaccessible to the antibodies, and the tuberculous process goes on uncured or unaffected.

In the case of scarlet fever or measles and other severe infections we presume that it is a struggle between the antibodies and the invading bodies. These antibodies, by their digestion or through their combat with the invading organisms, liberate a toxic product, but finally the antibodies overcome the invading organisms and the disease comes to an end. Not so with tuberculosis. The organisms have gained access to the bronchial glands, the bronchial glands begin to enlarge, become infiltrated, and then produce symptoms, possibly by reason of mechanical pressure or possibly by reason of their inflammatory character. These babies now show temperature curves and, in addition to this, a peculiar brassy cough. The cough, as I mentioned, is paroxysmal, with an expiratory crow. As the condition continues other pronounced symptoms may develop. For example, by reason of pressure on the bronchi evidences of bronchial stenosis may occur, or by reason of pressure on the recurrent laryngeal nerves laryngeal paralysis may ensue, with

a very hoarse cough; or there may be pressure on the veins, producing symptoms of suffusion; or sometimes these patients may have spinal pains because of pressure on the spinal nerves; and occasionally by auscultation we obtain a peculiar humming sound over the sternal notch—the Eustace Smith sign. Further physical signs which we may emphasize are those of a bronchitis. Sometimes an area of dulness may be elicited between the scapulæ, or not infrequently, by pressure of the enlarged glands on the bronchus, there may be alterations in breathing—the breath sounds may be diminished or may be bronchial in character.

There is another clinical type. A bronchial gland tuberculosis may be the focus from which other portions of the body may become invaded. For example, the lung may become involved through the blood-vessels or lymphatic channels. Pulmonary tuberculosis may manifest itself as a caseous pneumonia which may run a rapid course with all the clinical manifestations of pulmonary tuberculosis, with great wasting, anemia, and continuous fever. These are three points that we observe in the symptomatology of tuberculous processes wherever they are. Or we may have another manifestation, namely, a generalized bronchopneumonia in contradistinction to a caseous pneumonia, and the diagnosis of a bronchopneumonia tuberculous in origin from one non-tuberculous is sometimes very difficult or almost impossible.

The pulmonary affections of infants are not so characteristic and differ greatly from those of older children and adults. As a general proposition, it may be said that the younger the child, the more acute is the course of a tuberculosis. During the first year of life pulmonary tuberculosis may present itself under the clinical course of an acute bronchopneumonia, causing death in a few weeks. Pathologically, we know that toward the end of the first year healing processes tend to occur in the lungs. It has been shown that small cavities may occur in the lungs of infants, though hemoptysis is rare. The process occurs less frequently at the apex than in adults, and is found scattered throughout the lungs.

These infants give the impression of being acutely ill. They

are markedly prostrated, they cough, have the characteristic expiratory crow, run a febrile course, and the breathing is dyspneic. The tuberculin reaction is usually positive, though the bacilli are seldom found in the sputum. After the first year, in the "runabouts" in the kindergarten period and during the first school age, the localization of the lesion is extremely difficult. These children very often are pale and thin, possibly have a slight febrile reaction, though the physical findings are frequently negative. Even the Röntgen ray examination may be misleading because other pulmonary lesions may simulate tuberculous processes.

Starting at the tubercular bronchial gland the tubercle bacilli may invade the body through the blood-stream, and a disseminated miliary tuberculosis may result. Now, acute miliary tuberculosis may manifest itself in a variety of ways. First, we may have a cachectic type with a slight elevation of temperature, irregular in character, constant loss of weight, slight glandular enlargement, a slight enlargement of the spleen, some thyroid or thymus enlargement, and abdominal distention. The baby is losing weight continuously, is suffering from what is supposed to be a marasmus, and failing steadily. Possibly at the end a localized tuberculous process manifests itself or possibly the baby dies from exhaustion without any palpable or evident localization, and at the autopsy, if one be made, it is found that the infant has died from an acute miliary tuberculosis whose clinical manifestations consisted of fever, great anemia, and exhaustion. That is the cachectic type—frequently not recognized until after death.

In another type a baby falls ill with fever which runs a constant course. There are no focal findings of any importance; possibly the von Pirquet is negative because (as we will explain at another time) during an acute miliary tuberculosis, when the antibodies are used up, the von Pirquet reaction does not become manifest. The fever continues, the baby becomes more and more exhausted and more anemic, and possibly a little later some pleuritic or pulmonary manifestations develop, or some abdominal form of tuberculosis or a meningitis will clear up all the seemingly obscure disease conditions.

Then we have to do with the pulmonary form of miliary tuberculosis. This form is usually characterized by the presence of a large number of tubercles in the lung, though caseous foci rarely occur.

How do we diagnose tuberculosis in infants? We have already referred to the diagnosis in the bronchial gland type. Clinically, by the *x*-ray, by the increased area of dulness in the interscapular space, altered type of breathing, and the other clinical symptoms previously mentioned.

The pulmonary types are difficult to distinguish from the non-tuberculous affections. Then, too, the conditions that are so common in adults which indicate tuberculous processes are not present in infants as a rule, and do not aid in diagnosis as they do in older individuals. In infants particularly, and especially in the acute miliary types of tuberculosis, the greatest aid to diagnosis is the tuberculin test.

As you know, the tuberculin test of von Pirquet consists simply in placing a drop of tuberculin on the skin and then making a slight abrasion, and noting the reaction which occurs within twenty-four to forty-eight hours. This reaction occurs first of all in a flush or redness of the skin, then a slight papule appears with a red areola, accompanied by some induration. At the end of forty-eight to seventy-two hours this begins to recede. At first the color is a bright red, then tends to become darker and more of a terra-cotta or copper tinge, and then fades gradually into the normal color of the skin. This reaction varies in intensity. Let me say here that in the technic only a slight abrasion, using no considerable force, is necessary. Sometimes vesicles form at the site of the inoculation; sometimes a marked induration occurs with a very large or very marked areola.

I have already said that in some cases of tuberculosis we get no reaction at all from the von Pirquet test. In the case of an acute miliary tuberculosis it is a well-known fact that no reaction occurs, and, as I have attempted to explain, it seems that the antibodies are all consumed or exhausted, and, consequently, no reaction occurs. In measles the von Pirquet reaction is quite generally absent.

There are other tuberculin tests. The so-called Moro test is almost the same, except that the tuberculin is contained in an ointment (lanolin); it is rubbed into the skin, and within twenty-four to forty-eight hours a dermatitis occurs at the site of the inunction. There is the Calmette test, which consists in dropping tuberculin on the conjunctiva, but for our work this test has no value. During the early days, when this test was tried occasionally, some children lost their sight, and, particularly when there was some latent tuberculous process about the eye, it was likely to become active. We use Koch's old concentrated tuberculin here entirely.

The intracutaneous test is sometimes employed, and consists in the injection of tuberculin between the layers of the skin with a very fine needle. The tuberculin is used in a solution of 1 : 50,000, 1 : 10,000, and 1 : 5000 in normal salt solution. With this test an obscure tuberculosis is often brought to light. As little injury as possible should be inflicted on the tissues in making this test.

Now, so far as other methods of diagnosis are concerned, we lay great stress on the Röntgen ray in cases of bronchial gland tuberculosis, pulmonary tuberculosis, and bone tuberculosis. We must remember that the diagnosis is not always cleared up by the *x*-ray alone. Because there is evidence of consolidation in the lung we cannot assume in every instance that it is tuberculosis. Other disease conditions may give similar pictures. The *x*-ray picture, if taken in connection with the clinical course and symptoms, will prove a valuable link in the final judgment as to the condition of the patient.

Sluka describes the Röntgen findings in hilus tuberculosis. He finds a triangular shadow, the base of the triangle being imposed on the heart and blood-vessels and the apex extending into the pulmonary field. He finds the condition more frequently on the right side than on the left. On the right side three different forms were observed: a triangular shadow, a semicircular shadow with the convexity upward, and a semicircular shadow with the convexity downward. On the left side two forms were observed: a shadow reaching from behind

the heart toward the periphery, with a convexity either upward or downward.

Sluka concludes that hilus tuberculosis in children can be easily overlooked, and that in the early stages it can be recognized only by the Röntgen rays.

INFANTILE TUBERCULOSIS (*Continued*)

At the previous clinic we were discussing infantile tuberculosis. The baby that was shown then had what type of the disease? Bronchial gland tuberculosis.

Now again, in discussing the diagnosis of infantile tuberculosis, we shall consider the clinical course and the clinical signs as reflecting the diagnosis. Remember we said that these infants with tuberculosis ran a temperature. There is nothing characteristic, however, about it; sometimes it is moderate, sometimes it is extremely high, and is constant as in typhoid. We referred to that in our clinical classification. We spoke of a typhoidal type. We may say that in a large number of acute cases of infantile tuberculosis fever is present, and then it follows no typical course, though it may be remittent in character.

Let us pause a moment here and consider the errors into which we may fall in considering fever. A young or imprudent doctor may come to the bedside of an infant and, on hearing the story of fever, suggest to the anxious mother that possibly the baby is suffering from tuberculosis. Now there are other conditions than tuberculosis that tend to produce elevation of temperature. For example, an obscure fever of infancy may be due to *colon bacillus infection of the kidney or bladder, or both,* and this is not an uncommon cause of continued fever in infants. So do not make your diagnosis of tuberculosis until you have examined the patient several times and tested the urine microscopically. By the way, what does the urine show in a colon infection of bladder or kidney? There may be an intermittent pyuria or a long-continued pyuria. What organisms

do we usually find? As a rule, the colon bacillus. We must not forget these simple things.

The baby may be suffering from some other cause of fever—*food disturbance.* How do you prove that there is food disturbance? Go into the history of the feeding. Some of these babies with febrile reaction may simulate tuberculosis. What shall we do? Remove the food. If you take the food away for twenty-four hours, remove from the diet everything that is likely to cause putrefaction or fermentation. If no food is being digested or the food is indifferent, the fever should disappear. We have already learned that; it is one of the things we laid so much stress on.

Otitis Media.—Infection in one or both middle ears may cause very little pain, and yet there may be an infection in these organs characterized by considerable fever, and sometimes running a protracted course.

Typhoid may occur in very young infants, and should at least be thought of in the process of differentiation.

Acute Endocarditis.—As far as endocarditis is concerned, in the majority of cases we can differentiate by the physical examination.

So we come to the conclusion then, so far as fever is concerned, that a very considerable number of diseases may simulate the febrile conditions in tuberculosis.

Another thing, and I think it is well to bear this in mind, is that infants sometimes have a slight febrile reaction that is extremely difficult to explain on a pathologic basis. There are babies that you visit from day to day and from month to month who will continually show a temperature of 99° F., 99½° F., and even 100° F. by rectum, and you really do not discover any pathologic lesion. I have watched such children grow up who were a source of anxiety to me. They are mostly neurotic children; children of highly organized parents; children for whom there was great concern displayed; mostly in families where the temperature was taken every day, where everything was painfully watched, and where the infants were nurtured and cared for as though they were "hot-house" plants. These babies frequently run a little temperature.

Now, if you have done everything possible to make an accurate diagnosis, and have exhausted every legitimate method of investigation, you may finally, by a process of exclusion, say, "Well, there is no pathologic cause for this febrile condition; it seems to be normal for this child." It works out that way many times.

Sometimes the *tonsils* or *adenoids* are the source of a chronic infection, and with their removal the cause of the fever disappears.

At any rate, referring again to the value of the temperature curve in the diagnosis of tuberculosis in infants, we may say that the majority of such babies have a temperature; that the fever is not typical, not characteristic, and, indeed, in some cases the disease may run its course without any considerable temperature.

The babies may lose in weight, and in some instances it is like the loss of weight in severe gastro-intestinal affections, such as the so-called decomposition. Loss in weight in tuberculosis may be precipitous or it may be very gradual. In some instances, especially in the older children where the infection is not active, there is no marked loss of weight. An infant's weight may remain stationary or, indeed, where the process is latent, the infant may gain.

Many infants cough; some have a paroxysmal cough. The cough that is characteristic of tuberculosis is the one associated with bronchial gland involvement. It is accompanied by an expiratory crow.

The *physical signs* of pulmonary tuberculosis in infancy are not altogether reliable. If, for example, pulmonary tuberculosis is of the bronchopneumonic variety, then we have nothing particularly characteristic of tuberculosis, except, possibly, the protracted course of the disease. Or if in another case it assumes the character of caseous pneumonia, we have the characteristic feature of a protracted, long-drawn-out, massive consolidation, without anything that is typical of tuberculosis except possibly the wasting and the long-continued fever. I have already called attention to the fact that cavity-formation in

infants is relatively rare. Though cavities may occur in older children, they do not commonly occur in young infants. Occasionally at the site of a tuberculosis an abscess may form. I may say, by way of repetition, that tuberculosis in the apices of the lungs is not common in infancy. This has been demonstrated for a long time. Some of the authorities of the past laid great stress upon it, and the difference between tuberculosis in children and adults consisted in the fact that in the latter the physical signs were most frequently found in the apices, whereas in the former they were confined to the lower lobes.

Some years ago Dr. Holt called attention to a procedure by which the sputum of infants could be obtained by exciting cough. As the child coughs and expectorates into the pharynx the mucus is collected upon a piece of gauze, washed in a glass dish, and spread upon a slide and stained for tubercle bacilli, and, indeed, he succeeded in finding tubercle bacilli in sputum collected in this manner.

X-ray pictures are of the very greatest value, in that they show consolidations and contraction of the pulmonary tissue away from the hilum; also show infiltration of the bronchial glands.

It has recently been emphasized that minute papular eruptions occur on the body about the size of a lentil, at first raised and indurated, of reddish hue; possibly later on they may be covered with scales, and soon the papules become brownish-red in color and eventually disappear. These papules must be carefully searched for; sometimes there may be one or two on the body of the infant; at other times there may be quite a crop of them.

There are other skin affections closely associated with infantile tuberculosis, such as those caused by the scrofuloderm, with its tendency to the production of superficial ulceration. And then there is lupus, which is rare in infancy.

There is still another lesion about which there has been considerable discussion, and which some writers would associate with tuberculosis, and that is erythema nodosum. It occurs

like this: First, the baby suffers pain for a few days (pain about the joints and pain over the shafts of the bones), then upon the extensor surfaces, usually of the arm and leg, some little plaques appear which soon look like hives and increase in size; they may grow as large as the palm of the hand and are raised; there is considerable fever; very soon these plaques become glistening and shining, and have a decided reddish-pink or rose-red hue. Often several large plaques will coalesce and form larger ones. Usually you will see them below the knees. The lesions disappear gradually. This eruption has been observed in children with active and latent tuberculosis. It has been noted, too, that some children who have been afflicted with erythema nodosum may later on tend to develop an active or acute tubercular process to which they succumb. I have seen such infants suffer from tuberculous meningitis or other acute tuberculous processes which ended fatally. Those who have searched the literature extensively will find that occasionally an erythema nodosum is followed by tuberculous processes. The relationship between erythema nodosum and tuberculosis may be a mere matter of coincidence, and a pathologic connection between the two diseases is not definitely settled.

Then another condition to which I call your attention is *phlyctenular ophthalmia*. What does it look like? On the conjunctiva or on the cornea there will be noticed one or more vesicles with a circumscribed zone of inflammation, the most pronounced symptom being a photophobia. Eczema is commonly associated with this disease. It occurs more often among the children of the neglected classes; it is particularly prevalent in unhygienic surroundings; you see it more commonly in your poor practice than among well-to-do people.

The question that arises in our mind all the time is whether or not these phlyctenulæ are a manifestation of tuberculosis. There has been a good deal of discussion among oculists as to their being characteristic of tuberculosis. If one analyzes carefully all of the signs and symptoms, the physical findings and the related conditions, one is forced to the conclusion that there is not sufficient evidence to justify the belief that phlyctenular

ophthalmia is an absolute indication of tuberculosis. It may be a concomitant condition. The same improper hygiene, the same uncleanliness, the same debilitating conditions which produce tuberculosis may in other cases produce phlyctenular ophthalmia. In some of the cases of phlyctenular ophthalmia the von Pirquet is negative, therefore we might say that the disease occurred in the absence of tuberculosis. We know, on the other hand, that in a large percentage of cases with latent tuberculosis the von Pirquet is positive after the third or fourth year of life; consequently, we say that the evidence is not sufficient for us to assert that phlyctenular keratitis is an absolute manifestation of tuberculosis. The two conditions may be present in the same individual, the one not depending on the other. Let us consider for the moment the lymph-node in the diagnosis of tuberculosis. We know that in examining an infant we pay particular attention to the enlargement of the solitary lymph-nodes. Very often in an infant or even in older children (third or fourth year) we find enlarged lymph-nodes, and we are confused as to their nature and significance. Not every enlarged lymph-node is tuberculous. There is at present a mild epidemic of a condition that is characterized by high fever and glandular enlargement. Recently a lady came to me from a near-by city with her three-year-old child, who had been suffering from swelling of the lymph-nodes in the anterior cervical region. The swelling was bilateral, there was some edema, and moderated pain in the affected region. Fever was constantly high, the child was prostrated and pale. There were no marked findings in the pharynx or nasopharynx, and in the course of conversation the mother related that there were many similar cases in her city. In the course of two weeks the fever had disappeared and the glands had become much diminished in size. In view of the epidemic character of the disease, high fever, and the acute course, we concluded that the condition was one of glandular fever, also known as Pfeiffer's disease.

Adenitis of the neck follows various affections of the teeth, mouth, gums, nares, tonsils; also the exanthematous fevers, diphtheria, measles, and scarlet fever.

In localized eczemas and mild skin irritations the adjacent lymphatic glands become enlarged.

Still's disease is a condition of arthritis deformans characterized by an enlargement of the joints and the adjacent lymph-nodes, and usually accompanied by fever. The nature of this fever is still under discussion.

In Hodgkin's disease multiple glandular enlargement occurs, associated with profound anemia, and the differentiation is sometimes extremely difficult.

Tubercular lymph-nodes may occur in the cervical region. They are multiple, they tend to coalesce into larger masses, and eventually to become adherent to the skin. The course is chronic; they may terminate in resolution; more often in caseation or in abscess and sinus formations.

The lymph-nodes in the supraclavicular region when palpable are nearly always suspicious of an underlying positive tubercular pulmonary process, and should be regarded seriously.

Prognosis.—We come next to the prognosis of infantile tuberculosis. What becomes of this large number of tuberculous infants? It is said that the younger the infant, the graver the prognosis. Children under six months of age present an unfavorable outlook, and only a slightly better prognosis up to one year. And still, with our refined methods of diagnosis, with our skin-tests, and with our intracutaneous reactions revealing almost every form of latent tuberculosis, I should advise you to be somewhat cautious in making an absolutely unfavorable prognosis. Indeed, it has been my own experience in some cases where I pronounced the doom of an infant because of a positive tuberculin reaction with fever and slight cough, that the baby grew fat, happy, and prospered. Not all infants who present a positive tuberculin reaction are necessarily doomed. It is true that the infant is in great danger, but it is not true that the infant with tuberculosis need necessarily die.

In children of the third or fourth year the disease becomes more common; it is more likely to be latent, and hence the disease is less likely to be fatal. This suggests to us the hypothesis that Behring introduced into the subject of tuberculosis.

He said that nearly all tuberculosis was contracted in the first period of life during very early infancy, and that in a large number of cases this tuberculosis was an ingestion tuberculosis, possibly through the medium of milk; that it affected the mesenteric glands and there it remained latent; later in life, at puberty, or the period of growth or adolescence, or even in adult life, this tuberculosis became manifest and involved the lungs or bones or joints, or other organs; in fact, that it was latent or inactive in the body of the individual for many, many years.

To summarize the points in the prognosis of tuberculosis, we may state that tuberculosis in infancy is rather unfavorable to the life of the baby, but that, nevertheless, the prognosis is not absolutely bad; that in older children tuberculosis may remain latent for years, and in many cases may become healed without offering any meance to the future health or the life of the individual.

The next point for our consideration is the prophylaxis of tuberculosis. How shall we prevent tuberculous infection? The prevention of tuberculosis in infancy follows from what has been said.

An infant who is born of a manifestly tuberculous mother should be taken from the breast as soon as possible. Indeed, we learned earlier in the year that an actively tuberculous mother should not nurse her infant under any circumstances. Then, if possible, an infant should be taken out of a tuberculous environment; children should not be raised in families where there is a tuberculous relative. People will frequently come to you and ask if it would be advisable or safe to go away to the mountains or the seaside with the baby and its mother and take along an old aunt or a dear relative who is a little tuberculous. Frequently that happens. The only answer is that the infant must not be in the same environment with one who has active tuberculosis.

Nurses and nursery maids are frequently responsible for conveying tuberculous infection in families where great care is shown for the health and life of the baby. The nurses who care for these babies should be subjected to medical inspection.

Dr. Alfred Hess, of New York, tells of the case of a woman, an infant's nurse, in charge of an institution or a number of beds in an institution who conveyed tuberculosis to several babies because she herself had the disease. We know of a nurse with an open tuberculosis who went about the city here taking one situation after another, and she undoubtedly conveyed tuberculosis to several infants that we know of.

Infants develop respiratory tuberculosis, bone tuberculosis, and bronchial gland tuberculosis. The assertion cannot be too strongly emphasized, therefore, that an individual with an open tuberculosis is a menace to another person, especially to a young infant. Those who have followed the literature will remember a paper by Dr. Fishberg, of New York, giving a résumé of his investigation of the lower part of the East Side in New York. He studied the environment in particular, and found that children of large families, living in rather close quarters with adults who had more or less manifest tuberculosis, presented positive reactions in a larger number of instances than those who were brought up in better surroundings. So it is the world over. Infants reared in crowded quarters under bad hygiene, subjected to the unfit living conditions that obtain, are likely to become tuberculous.

It is not, however, only a question of environment; it is not only a question of open tuberculosis; it is also a question of foods. There is no doubt in my own mind that tuberculous food may under certain circumstances cause primary intestinal tuberculosis. It is true, and we must accept it as proved, that the largest number of cases recorded show that tuberculosis is bronchogenic in origin; but, on the other hand, there is a certain class that is enteric primarily, and this is probably due to the bovine type of the bacillus. For this reason, then, milk should be controlled, and in all doubtful cases should be pasteurized.

There are other methods of conveying tuberculosis. For instance, in New York a few years ago Dr. Holt reported circumcisional tuberculosis. This is extremely rare.

In other cases midwives have conveyed tuberculosis. There is a well-known case of a certain midwife who conveyed tuber-

culosis to such an extent that almost every baby she delivered showed signs, and on investigation it was discovered that this woman employed mouth-to-mouth resuscitation.

The treatment of tuberculosis in children is like the treatment of tuberculosis in adults. First of all, it is dietetic and hygienic.

Let us speak of the dietetic first. We should feed these babies on the most rational plan possible. We should give a well-balanced diet that they can digest and assimilate.

Schlutz, of Minneapolis recently showed that a diet that ensures the most fat deposit in the tissues is the most decidedly beneficial to the tuberculous child.

The use of carbohydrates is advisable, notwithstanding the fact that Czerny taught that when carbohydrates are given in excess there is water retention in the body. Water retention, says Czerny, tends to diminish immunity against the tubercle bacilli and their products. This statement lacks confirmation. Infants may be given a carbohydrate diet. In other words, babies may be fed to the maximum; overfeeding should be as carefully avoided as underfeeding.

From the standpoint of hygiene, babies should be brought up under the best possible living conditions. The infants in the poorer districts should receive the attention of the social workers and visiting nurses; they should live in the open air as much as possible; the home should be ventilated thoroughly.

Then comes the question, Should these infants be taken away? Children should be taken to the open places when the weather permits. Older children may be sent to forest schools or open-air schools, or infants and children may be sent to the seashore or the mountains.

Rollier, at Leysin, you remember, has shown to the world how much can be done by heliotherapy. His patients there on the Swiss mountain peak are exposed continuously to sunlight, and the photographs we see from this resort show that children in the midst of winter are running about exposed to the sun, very lightly clad; indeed, they are seen going through the snow with a minimum of clothing. The fact of the matter is, that in this

mountain resort Rollier has obtained the most remarkable results in tuberculosis of the bone, joints, skin, and even in cases of pulmonary tuberculosis. He has shown that the action of the fresh air and the direct sunlight are the most powerful agents in the treatment of tuberculosis. We are told that Professor Kocher prefers to send his surgical cases to Rollier rather than treat them himself by operative measures. Patients remain in the resort for several years exposed to the radiant sunlight, with most remarkable results. Rollier's statistics of cures are simply overwhelming, but true. Men who go there are enthusiastic over the results. So it is probable that the same method could be employed everywhere, though perhaps not quite so successfully. We have sunlight some days everywhere and fresh air all the time, unless it is polluted with dirt and soot. So much, then, for the hygienic treatment. Food, fresh air, ventilation, the seaside or mountains where the children can be sent.

How about medical treatment?

Young infants often do well on cod-liver oil. There is no specific medication. Sometimes they improve on iron and arsenic where the anemia is severe. As a general rule, we must say we get no results from drugs.

How do we stand on tuberculin? I think somebody has said there are 127 different kinds of tuberculin; they have all been tried, and at the present writing there are fewer enthusiasts about the use of tuberculin than there were a few years ago. So far as the statistics and the results of other men are concerned, and so far as our own results are concerned, we are not convinced that the curative results are conclusive; so that at the present moment we do not feel that we have the real specific tuberculin treatment, and consequently we cannot advocate or teach it.

SARCOMA OF KIDNEY IN A CHILD EIGHTEEN MONTHS OLD. FREQUENCY OF NEOPLASMS IN INFANCY

THIS morning we have a little patient with this history. (History read.)

We have here, then, a baby one and one-half years old, undersized, considerably emaciated, and pale; the skin of the face is covered with eruptions, the mucous membranes are not pale. There is some cracking of the lips covered with dry crust. The child has erupted twelve teeth and the cavity of the mouth seems normal. The pupils are normal; there is no rigidity in the neck. There is a slight enlargement of the lymph-nodes in the posterior cervical region; a few nodes may be felt at the angle of the jaw, particularly on the left side. The skin has lost its elasticity to some degree. Note that if elevated the skin does not snap back into position. The musculature is soft and flabby. There is nothing abnormal about the external contour of the chest, and the examination of the thorax by percussion is normal; auscultation over the lung reveals normal vesicular breathing; the heart tones are normal. There are no deformities about the chest, no evidence of rickets, either in the thorax or in the extremities. There is no dilatation of veins on the surface of the abdomen and no superficial edema. On inspection you will note a slight distention in the left upper quadrant, and there is a fulness here, and if you will compare the two sides you will note an asymmetry caused by an increase in size of the left upper portion of the abdomen; and if you will examine more closely you will see a nodular protuberance which is raised and rises above the level of the mass. It is the size of a twenty-five-cent piece, and it moves up and down with respiration, the nodules standing out like buttons. Percussion of the right side of the abdomen shows that the liver occupies its normal position, and there is no dulness; and, indeed, no painful area over the hepatic areas. When we come to examine the left side of the abdomen we detect dulness on percussion, which continues downward to a transverse line which lies on and

below the level of the umbilicus. Below this line we find normal tympanic resonance.

Percussing in the opposite direction, we note that the dulness extends from the midabdominal line in the inguinal regions on both sides to the anterior axillary line. There are a few enlarged lymph-nodes. There are no abnormalities about the extremities.

Now, coming back to the mass in the abdomen: As we palpate we find that the mass is freely movable. We are pleased to note this, as it indicates that no extensive adhesions have formed to contra-indicate surgical treatment, and grasping it, so to speak, we estimate that it is about the size of a fetal head. It gives the impression of being a spheric mass and its surface is nodular; its edges are plump and rounded. It is slightly tender on deep palpation. We must not palpate too violently, because it is possible to traumatize the tumor mass and produce a severe hematuria; or, indeed, it is possible that tumor masses may be dislodged, giving rise to metastatic processes. We now examine the mass bimanually, and find that it may be felt distinctly approaching the examining hand in the lumbar region; also that by pressure on the posterior aspect of the mass it may be felt to approach the examining hand on the anterior surface of the abdomen.

Now what have we here?

VISITOR: I think it is the spleen.

DR. ABT: I want you all to feel it. Dr. Lind, did you make a blood examination in this case?

DR. LIND: The blood examination showed a normal count of leukocytes and red cells. There were no morphologic changes.

DR. ABT: Now let us have some suggestions as to the diagnosis in this case. Or let us put it in another way. What are the possibilities? Let us first talk about the organs. The spleen has been suggested. Indeed, in this location the spleen cannot be left out of consideration. The tumor which we feel is spheric. There are no sharp edges. The mass, though easily felt, does not lie as superficially as it should if it were an involvement of the spleen. We inflated the bowel and found that the distended

bowel occupied a position in front of the mass. This speaks against a spleen.

This mass is nodular, indicating unevenness of growth, possibly a malignant growth. Malignant neoplasms of the spleen are very rare, though, of course, they may occur. Steffen searched the literature and found only 4 cases of malignant neoplasm of the spleen. In splenic tumors, such as occur in Barti's or Gancher's disease, a leukopenia would be of great diagnostic importance, while a leukocytosis and morphologic changes would be of great importance in diagnosis. It may be stated, too, that the kidney tumors are by far the most frequent of all abdominal growths in children. This is not a valid diagnostic reflection if alone, but considered in connection with all the other facts and findings it is a point which may be used in arriving at a judgment in a given case.

We may dismiss without discussion tumors of the bowel and tuberculous masses resulting from tuberculous meningitis, and consider that we have before us a tumor of the kidney. If this is a tumor of the kidney, what is its nature? There are a variety of conditions that we must think of. A congenital malformation of the kidneys, such as horseshoe kidney, where both are fused into one mass.

I recall a case where a baby a few weeks old was brought for examination, and we felt a tumor mass in the midline of the abdomen, lying upon the promontory of the sacrum. The urine contained abundant pus and the baby was running an irregular fever. Autopsy showed a fused or horseshoe kidney, with a secondary pyelitis and pyelonephritis.

Not infrequently hydronephrosis occurs in infants and children; it may be congenital or acquired. Double hydronephrosis is usually due to some congenital deformity, and death usually takes place before fluid accumulates and tumor formation takes place. In unilateral hydronephrosis the tumor mass may become of immense dimensions, filling up a great portion of the abdominal cavity. The tumors are smooth and rounded, but may be irregular in outline, not nodular, however. It may fluctuate and have the consistency of a cyst. It is quite char-

acteristic of these tumors that they vary in size from time to time. The symptoms may be due to the mechanical effects of the tumor.

I think you will all agree with me that our patient under examination is not a case of hydronephrosis.

Congenital cystic degeneration of the kidneys is one of the conditions which is encountered in young life. This condition usually occurs in both kidneys, sometimes so large as to render birth impossible. Though bilateral multiple cystomata are found on autopsy in older children, we showed you a case of unilateral cystic kidney earlier in the year in a little girl aged four years. The degenerated kidney was removed and she made an excellent recovery. The unilateral cystic kidney is rare, and only a few cases are reported in the literature.

Malignant tumors, especially sarcomata of both large and small intestines, may occur in early childhood. Tumors of the intestines are frequently taken during life to be growths connected with the kidneys, the omentum, the ovary, the peritoneum, or they may simulate appendicitis; or, owing to metastases, they may lead to the formation of multiple tumors; this makes the diagnosis of the primary lesion very difficult.

Pelvic tumors in children are rare, though dermoid cysts and simple cysts of the ovaries may occur. Sarcomata of the ovary occurs rarely. Rosenstein reports a case of carcinoma of the uterus in a two-year-old child. It is a common error to mistake a large tuberculous deposit in the loin for a kidney tumor. The correct diagnosis in such instances is frequently not made until the abdomen is opened.

It is not at all uncommon, if one examine carefully, to find kidneys that show abnormal mobility, and occasionally these movable kidneys will become markedly enlarged. For instance, I remember very well one of our cases that we had here in the hospital some years ago, a child of five or six, in whom we were able to demonstrate a wandering or a very markedly mobile kidney. Occasionally when the kidney was found too near the pelvis the ureter became twisted, and the child would be seized with attacks of violent pain resembling renal colic. It was neces-

sary to give sedatives in order to relieve the suffering until the kidney could be replaced and held securely by some form of bandage. These cases are found more frequently in girls than in boys, and the right kidney is involved more often than the left.

Tumors of the kidney may be benign or malignant. Among the benign tumors lipomata, fibromata, and fibrolipomata are most common.

But now, on the other hand, we have a number of tumors of the kidneys that are very common in childhood, especially in early childhood; they are malignant in character, and these tumors you know are carcinoma, sarcoma, and hypernephroma. The sarcomata are probably the most common.

I quote from a book written by Prof. Steffen, of Stettin, in which he cites a great many cases from the literature; he reports 219 cases of renal tumors in children of various ages. One is in a baby five months of age, a carcinoma of the right kidney that increased very rapidly in size; the tumor was the shape of a large globular mass that caused pressure upon the abdominal contents to such an extent that it resulted in the death of the child at the age of eight months. Convulsions occurred at the close of the illness.

I am just selecting a few of these cases. (Turns pages of book.) Here is another child in whom a primary kidney sarcoma occurred in the ninth month of life (a little girl). Then there is another group of cases that I want to mention. Here was a case of congenital sarcoma of the liver in a child of three weeks, which very rapidly increased in size and involved nearly all of the abdominal viscera, including the kidneys.

Here is a case of fetal kidney cancer occurring on the left side. The fetus was born dead. The left kidney was nearly twenty times as large as the right, and the carcinomatous infiltration invaded the cortical substance of the liver and also the capsule of both liver and kidney.

So we find these conditions at all ages; they occur in very young children and even in the fetus.

Now let me call your attention to the prepared table made by Hirschsprung, who found in children up to one year 6

cases of kidney tumor; two to five years, 17 kidney tumors; five to ten years, 6 kidney tumors; eleven to fifty, 33 cases. More occurred during the first ten years than occurred during the rest of child life.

Rohrer found in 107 cases of kidney tumor in children that 37 cases occurred from birth to the tenth year; 4 between the ages of ten to twenty years; 10 between the first and fourth years; 6 occurred after the fifth year.

The point is, then, that malignant tumors are extremely common in young children.

What are the symptoms?

First we palpate the mass. The tumor, depending upon the nature of the growth, may be nodular. These tumors grow very rapidly. The children usually have a little fever and they may or may not have blood in the urine. Occasionally they have intermittent hemorrhages.

Most of them show marked emaciation, great loss in flesh, and a great loss in general activity, so that the older child will eventually take to bed. Then, too, a good many of them have pain; indeed, so severe that it is necessary to give them narcotics.

So far as the differentiation is concerned, we learn a good deal from our experience. We know that the largest number of these cases that we see are on the left side; that has been our experience here. We know, too, that occasionally we made a mistake in thinking that the tumor was the spleen. Even the kidney tumor may be flattened, and we may be persuaded to think that it is the flatness of the spleen; then, on further examination, or after surgical operation or at postmortem, we nearly always find that the tumor was the kidney.

This infant that we have here this morning is emaciated; the tumor mass is very large, apparently nodular, irregular in outline. The general nutrition of the child is disturbed, and we feel that it is probably a malignant growth. As to differentiating clinically between hypernephroma, sarcoma, and carcinoma, that is almost impossible. Indeed, it is sometimes extremely difficult even upon histologic examination of the tumor to make accurate diagnoses. The sarcomata are frequently mixed in type.

Indeed, the type of the sarcoma varies greatly, depending upon the portion of the kidney from which the section is taken. So it is almost asking too much to differentiate the nature of the malignant tumor clinically. We feel, however, that we have a malignant neoplasm of the kidney. The treatment of these cases is altogether operative. This child will be operated upon this morning by Dr. Emanuel Friend.

A good many of these cases, especially when the tumor has existed for a long time, assume an excessive growth. When the tumor is extremely large and very adherent the operation is extremely difficult, almost impossible. In the early stages of tumor development, when the mass is still movable and free from adhesions, there is some possibility of success. Indeed, there are a number of cases on record where successful operations have been done. Metastases do occur in these cases, particularly metastases of the lung, and at other times they are more remote, occurring in the bones and distant parts, or the tumor may involve both kidneys.

So the treatment is surgical if the case is seen sufficiently early and the tumor is free from adhesions. I have here a number of references to the literature; cases are reported every year, and the remarkable thing is that these kidney tumors occur not only in very young children, but even during fetal life.

I think we will stop at this point so that you may go up stairs and see the operation.

Operation by Dr. Emanuel Friend

Dr. Emanuel Friend: The case has been seen by the entire staff of pediatricians of the Sarah Morris Memorial Hospital, and the diagnosis of splenic enlargement or a kidney enlargement made, favoring that of an enlargement of the kidney, with probabilities of a sarcoma of the kidney.

The differential diagnosis of the case in hand lies, as Dr. Abt has suggested, between sarcoma, carcinoma, hypernephroma, congenital cystic kidney, hydronephrosis, and pyonephrosis. Sarcoma of the kidney is more common than carcinoma. It is

met with most frequently in children and in young adults. The growth of the tumor is rapid, and the tumor usually reaches an enormous size before it destroys life. The mass is smooth and pseudofluctuation is generally present. The tumor is composed usually of round cells. The malignancy is very great.

Diagnosis.—The diagnosis of sarcoma of the kidney is usually not very difficult. The only affections for which it is liable to be mistaken are hydronephrosis, pyonephrosis, carcinoma, and hypernephroma, and in adults on the right side a distended gall-bladder. Hemorrhage from the kidney in sarcoma occurs frequently, and its occurrence in children is very suggestive of malignant disease of the kidney. The retroperitoneal location of the tumor can be determined positively by inflation of the colon. If the tumor is intraperitoneal, it will be displaced by the distended colon; if it is retroperitoneal, the tumor can be felt less distinctly in front, and, where dulness existed before the inflation, there is resonance due to the location of the distended colon in front of the kidney. Soft sarcomata of the kidney present pseudofluctuation on palpation, and if a large cyst occupies the anterior surface of the kidney true fluctuation can be felt. In hydronephrosis and pyonephrosis, if any doubt exists between these affections and sarcoma, an exploratory puncture through the lumbar region is harmless and enables one to make a positive diagnosis.

Girls appear to be more predisposed to primary sarcoma of the kidney than boys. The tumor is composed of spindle and large and small round cells. The origin from a matrix of embryonal cells is well shown in sarcoma of the kidney by the frequency with which striped muscular fibers are found in the tumor. Of course, the urinary findings are of great importance in differentiating from pyonephrosis and the various true tumors of the kidney.

In cases of a large tumor mass involving the kidney, or where it is questionable from what organ the tumor arises, or where there is no other method, especially in young children, of determining whether or not the opposite kidney is present, it is well to do the transperitoneal operation.

(Incision made.) It is ridiculous to attempt to take this mass out posteriorly on account of its size and the small lumbar space.

The abdomen opened by a right pararectus incision. The tumor mass, which was about the size of a large orange, presented itself posteriorly to the posterior peritoneum. Exploration was made for the presence of the opposite kidney and it determined. An incision was then made through the posterior peritoneum covering the tumor mass and the kidney, separated from its surrounding attachments, and brought out of the abdominal wound. The vessels and the ureter isolated and tied separately by means of chromic gut. The tumor mass involves mostly the lower pole of the kidney; it and the kidney are removed *in toto*.

After removal of the kidney with the tumor mass and careful hemostasis the peritoneum was again placed in its former position. In children, especially in making abdominal incisions, one must be exceptionally careful, so that by coughing or by exertion they will not break through the stitches.

The peritoneum was then closed by means of three layers: first, plain catgut for the peritoneum; second, chromic gut for the fascia; and, third, a wax silk for the skin.

That was not a difficult case to do. In children the separation is easy. The operation lasted forty-five minutes.

(PATHOLOGIC DIAGNOSIS OF SPECIMEN 7462)

"Microscopically, this tumor exhibits great heterogeneity of structure in closely adjacent areas. The predominant picture is that of adenoma-like tubules arranged in irregular nests and whorls. The cells are columnar and of more or less atypical arrangement in various areas, in places breaking through the basement membrane and heaping up or forming solid masses, in other areas presenting the structure of a benign adenopapilloma. These areas are well, though not excessively, vascularized with vessels whose walls consist usually of only a layer of endothelium and a small amount of subendothelial connective tissue. Such areas are, of course, suggestive of the Wolfian anlage.

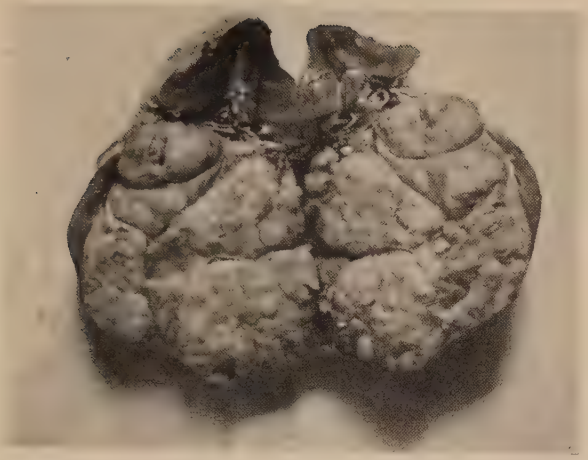

Fig. 18.—Kidney and tumor split open and showing incised surfaces.

Fig. 19.—Same, showing external aspect of kidney.

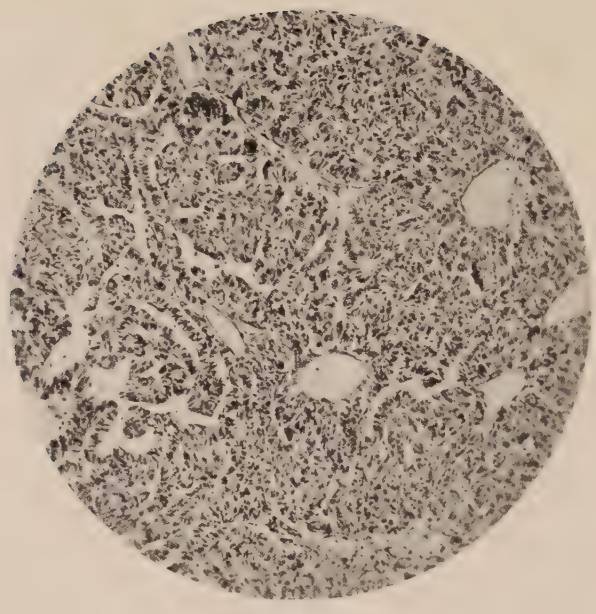

Fig. 20.—Detail from papilloma-like areas.

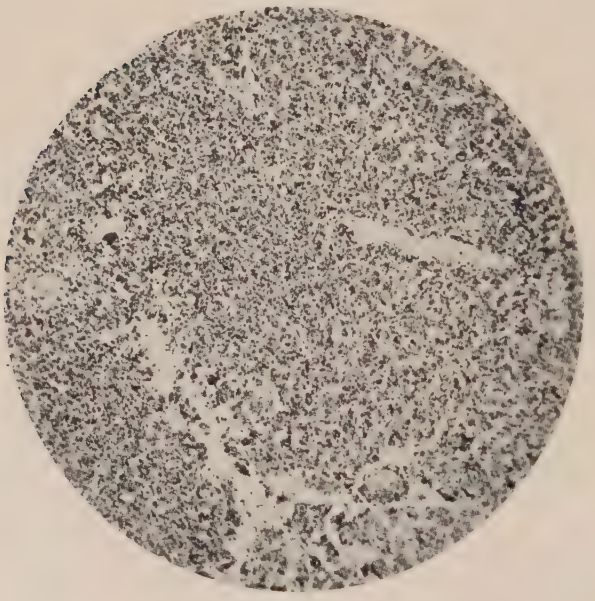

Fig. 21.—Detail from round-cell sarcoma areas.

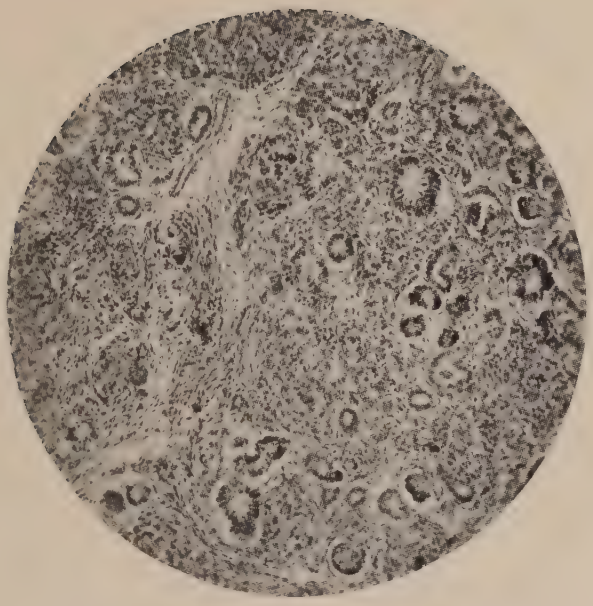

Fig. 22.—Detail from sarcoma areas, containing tubules and glomeruli.

'Other areas interpolated between those of the type just described have throughout the structure of an ordinary mixed-cell sarcoma, with predominance of small round cells, a few large round cells, and some slightly elongated (spindle) cells. Such areas are excessively vascularized with thin-walled vessels and display considerable interstitial hemorrhage. In certain of these areas these cells are so arranged with reference to large thin-walled blood-vessels as to suggest a hemangiosarcoma. (Such areas are characteristically found in this type of renal tumor.)

"Still other areas show the stroma of a round and spindle-cell sarcoma, in which, however, are numerous tubules resembling more closely those of the fully developed kidney, and also some glomeruli. Both tubules and glomeruli, however, depart considerably from the normal type, being hyperplastic to a greater or lesser degree. Thus, the tubules are sometimes solid, or their basement membrane line is ill defined.

"Diagnosis.—This is a typical specimen of the tumor described by Birch-Hirschfeld as 'embryonic adenosarcoma' or 'sarcomatous glandular tumor.' Perhaps it is more conservative to call it (with Kauffman) a 'congenital mixed renal tumor.'"

[NOTE.—I am much indebted to Dr. Edwin Kirk, Pathologist to the Michael Reese Hospital, for the pathologic report of the case, and to Mr. Merritt for the photographs.]

May 2, 1915: Wound healed by primary union, and child completely recovered.

CLINIC OF DR. ROBERT B. PREBLE

ST. LUKE'S HOSPITAL

CHRONIC LYMPHATIC LEUKEMIA IN A MAN OF SIXTY-FIVE, WITH A DISCUSSION ON THE CAUSES OF DYSPNEA

HERE we have a man who came in with this history: He is a blacksmith, sixty-five years of age, and he complains of cough and difficulty in breathing and some pain in the left side of the thorax. Two weeks ago he went to some mineral springs and took some baths, but became so much worse they sent him back to Chicago. The dyspnea got worse and he began to have some fever. The dyspnea is by no means such a short story. He had an attack four or five years ago, but it was not so bad that he had to give up his work. He has had more or less dyspnea since that time, but has never had any palpitation of the heart and never any swelling of the feet. He has had to get up two or three times at night to urinate, but that is no worse than it formerly was. There is no headache and no backache. About last November he noticed some swelling in the back and neck, which has been getting larger, but has not been painful or tender. He now notices some glands in the groin. What would you think about that?

VISITOR: He has a generalized adenopathy.

DR. PREBLE: Of how long a duration?

VISITOR: I should think of Hodgkin's disease.

DR. PREBLE: In the first place, we have a generalized adenopathy, and then, in the second place, what happened?

VISITOR: There is dyspnea.

DR. PREBLE: Is the dyspnea related to the adenopathy or independent of it?

VISITOR: I should say it was related to it.

DR. PREBLE: Yes, but the dyspnea has lasted for four or five years, and the adenopathy has lasted four or five months. How is that? (To Patient): Is your breathing much worse than it used to be?

PATIENT: Yes, much worse, and the last three weeks worse than ever.

DR. PREBLE: The dyspnea has lasted off and on for four or five years, but is distinctly worse, particularly in the last three weeks. How are we going to account for this dyspnea on the basis of his glands, if the glands only began four or five months ago and the dyspnea began five or six years ago?

VISITOR: The former dyspnea might be due to an emphysema.

DR. PREBLE: The intensification of his dyspnea within the last few weeks might easily be due to the pressure of the glands, but there are other factors which might give rise to the dyspnea which antedated the enlargement of the glands. Now, what could that dyspnea have been due to?

VISITOR: It may have been due to conditions in the chest, such as a chronic myocarditis.

DR. PREBLE: Yes; but in view of the fact that he has been a blacksmith all these years one might think it was an emphysema. It might be a cardiac dyspnea. (To Patient): Do you think the lumps have been getting any bigger since you first noticed them?

PATIENT: No; when they began there were only three, but the next morning there were more, and they were about as big as they are now.

DR. PREBLE: Where did they first show, in the groin or under your arm?

PATIENT: I don't know about that.

DR. PREBLE: We will remove the shirt and make some observations of the chest. What do you think about that cough?

VISITOR: It sounds like some obstruction.

DR. PREBLE: One of the curious things about him is that he wants to lie down; ordinarily, people who are as dyspneic as he is want to have their head elevated, but he wants to lie down; this is the first time I have seen him sitting up. The dyspnea is not always expiratory, and every time he coughs the dyspnea has with it that peculiar ringing, what does that suggest?

VISITOR: It suggests an obstruction.

DR. PREBLE: Now, as he sits up, what do you see? You note at once that he is dyspneic, fairly intelligent, wide awake, and you see the enlarged glands in the neck and in the axillæ; an obvious enlargement of the lymph-glands on both sides of the neck and in the axillæ; they are not so massive as one occasionally sees them, but still there is a very considerable enlargement and they are also enlarged in the groin. (To Patient): Are you comfortable that way? (Patient sitting halfway up.)

PATIENT: Yes, it's better for me.

DR. PREBLE: That's what I've been telling you, but you want to lie down.

PATIENT: I wish I could take a walk.

DR. PREBLE: Now that is about all that one sees on inspection, except that there has been practically no loss in weight. (To Patient): You haven't lost very much, have you?

PATIENT: I used to weight 225 or 240 pounds, but I don't weigh quite that much now.

DR. PREBLE: There has been no great loss in weight, no particular anemia, and, in spite of the dyspnea, no particular degree of ache in the chest. Catch this sound if you can; do you get any difference? Just below the clavicle on the right side, about $1\frac{1}{2}$ inches to the right of the median line, we get an area of dulness, and this area of dulness passes downward. On percussion we can outline an area of dulness which begins at the right clavicle and to the right of the median line curves downward and inward a little bit, and then passes down and joins the hepatic dulness to the left of the sternum. On the left it passes down, slightly curves inward, and joins with that of the sixth interspace, one inch beyond the nipple line. What could that area of dulness be?

VISITOR: Enlarged glands.

DR. PREBLE: It would naturally occur to one that the dulness was due to enlarged glands. Could it be due to anything else?

VISITOR: It could be due to aneurysm.

DR. PREBLE: Is there anything inherently improbable about it being an aneurysm? For example, if we refer that area of dulness to an aneurysm we will have to resort to a two-legged diagnosis, which we don't like. It is much more likely that that area of dulness is due to the enlarged glands. As a matter of fact, his aorta is dilated, as shown by the *x*-ray examination, but it does not constitute a very large portion of what the plates will show. It is, in all probability, due to the enlarged glands. What would you think about that?

VISITOR: I would think of tuberculosis or Hodgkin's.

DR. PREBLE: Yes, you would think of those things. Is there anything else? Would you think of syphilis?

VISITOR: Yes, but there are no symptoms. It might be leukemia.

DR. PREBLE: Leukemia? Remember he is sixty-five years old, and that is a little older than we usually find it, but they do occur at this age just as they occur in people much younger than twenty; but when you consider that leukemia is a rather rare disease, and when you take in addition to that his age, what would you think about it?

VISITOR: It would be easy to find out.

DR. PREBLE: Yes, the diagnosis of leukemia is a relatively easy proposition. As a matter of fact, he shows a very striking blood-picture—60,000 whites. Is that pathologic, is it necessarily a leukemia? 95 per cent. of those are small mononuclear leukocytes. What would you call it then? A chronic lymphatic leukemia? Why couldn't it be a whooping-cough? Here is an old man who coughs and wheezes and he has a leukocyte count of 60,000.

VISITOR: If it runs over 50,000 it is probably a leukemia· you don't get that in whooping-cough.

DR. PREBLE: Oh, yes you can. They sometimes run as high as that, and here is a man sixty-five years old; why couldn't it be that?

VISITOR: We don't get the adenopathy.

DR. PREBLE: That's it; we don't get the adenopathy. Whooping-cough would not account for the adenopathy. He has an enlarged spleen, not very large, about two fingers below the ribs. The liver I have not been able to feel. (To Intern): How about that big heart of his?

INTERN: There may be a secondary dilatation.

DR. PREBLE: The same thing happened as happens in any anemia, and we get degeneration from the anemia, but he is not essentially anemic. Here is a fellow who has worked hard all his life as a blacksmith, and the natural thought is that the enlargement of the heart is due to the hard work, a myocarditis. That's much easier than to refer it to a degenerative process that has been going on for only a few months. How about that leukemia; does it strike you as being right?

VISITOR: Yes, it does.

DR. PREBLE: Acute or chronic? He tells us that he found these glands about Thanksgiving time. If he found them then, it is likely that they were there two or three months before, so it possibly has been going on six or seven months. The lymphatic leukemias, as you know, have a tendency to run an acute course, but when we see one in an individual like this the course is usually chronic. The younger they develop a lymphatic leukemia, the more likely it is to run acutely. The older they develop it, the more likely it is to be chronic. They are much less common in a man of his age. One sees them, but they are exceptional, and to see a lymphatic leukemia in a man of sixty-five is rather an unusual thing. What are you going to do about it, anyway?

INTERN: Give him general treatment, keep him in bed, and, for the leukemia, I don't believe there is anything outside of the *x*-ray.

DR. PREBLE: Of course, we will take care of him, keep him quiet, and feed him, but, so far as the leukemia itself is concerned, the only remedy that offers anything is the *x*-ray. Benzol is much less likely to be effective in this case than in a splenomyelogenous leukemia, but we will put this patient on *x*-ray treatment and also give him benzol.

In a way the condition is a bad one because these glands are doubtless interfering with his respiration, and, in addition to the dyspnea of cardiac origin which we have, we have added a dyspnea from pressure, and one is uncertain as to how soon everything else will be lost in the dyspnea.

RENAL AND CARDIAC INSUFFICIENCY

THE patient I am presenting to you this morning has the following history:

Last November (about five months ago) this patient had an attack of bronchitis that was followed by shortness of breath and asthma, which condition lasted until about two weeks ago, when she entered the hospital. On account of the asthma she has to sleep in a chair. At that time her feet were swollen. There was also a puffiness about the face. Sometimes she would have a stupor lasting for six or eight hours. There was no headache. Was obliged to urinate several times during the night. She had nausea and vomiting for a number of weeks, with pain in the epigastrium. These attacks ceased two weeks ago. What do you think about this case? The patient is forty-two years old, and has worked right along up to within three weeks before entering the hospital. Her health began to fail last fall.

DOCTOR: There was no swelling?

DR. PREBLE: Yes, there was some swelling about the feet and face.

DOCTOR: Heart or kidneys.

DR. PREBLE: Yes, either heart or kidneys would account for the condition. Why?

DOCTOR: Because of the edema and dyspnea.

DR. PREBLE: Yes.

DOCTOR: It might be the liver.

DR. PREBLE: In what way?

DOCTOR: Cirrhosis. Or some lung process might cause it.

DR. PREBLE: What sort of a pulmonary process could give that combination of dyspnea and edema?

DOCTOR: A fibrosis, which would obstruct the circulation.

DR. PREBLE: That brings us back to the heart again. It is only rarely that a pulmonary process would produce such a condition. As a matter of fact, even the dyspnea is not well accounted for on the basis of a pulmonary process. Even where there is a great deal of destruction of lung tissue the patient rarely complains of shortness of breath. Tubercular patients (even those far advanced) rarely complain of dyspnea. It is only when the destruction of tissues is very rapid, or when the breathing surface is rapidly destroyed, that dyspnea is complained of. For example, in pleurisy with effusion, where one lung is gradually compressed, dyspnea is not common. If, however, the lung is thrown out of function by a pneumothorax, then intense dyspnea is present because of sudden reduction. But in most pulmonary processes destruction is so gradual that the patient becomes accustomed to it. It is only indirectly that the pulmonary processes give rise to dyspnea. Take, for example, emphysema. Gradually the right heart fails, and then these patients begin to complain of dyspnea. But it is caused in these cases by the failure of the heart. Therefore in a pulmonary condition it would be difficult to account for the dyspnea present in this case, and still more for the edema.

DOCTOR: Pernicious anemia?

DR. PREBLE: That condition could do it, but usually there are other phenomena which are so much more prominent than the dyspnea or edema—weakness, utter exhaustion, and so forth. When asked if they are out of breath they will say yes, but that and edema are not the striking things. What else would the features of that history suggest?

DOCTOR: Was there an examination of the blood-pressure?

DR. PREBLE: Yes. But what about the history given?

DOCTOR: It sounds like a nephritis.

DR. PREBLE: The general impression one gets from that history is rather referred to the kidney than the heart. What would the dyspnea continuing over such a long period mean?

Dyspnea caused by a kidney condition is usually of shorter duration.

DOCTOR: Was it toxic?

DR. PREBLE: Yes. Offhand, however, one would say that an individual who had dyspnea of toxic origin would either get well or get worse in less time than five months.

DOCTOR: It all depends on the persistence of the toxemia.

DR. PREBLE: I know. But one would say that a nephritis, giving rise to dyspnea severe enough to make the patient sit up all night and eventually quit work, would either kill the patient in less time than five months or the condition causing it would improve. And yet there are a few things that suggest that it is a nephritic case. The face, for example, is not compatible at all with cardiac trouble. Could the dyspnea of nephritis be due to anything else than a toxemia?

DOCTOR: It might be due to an associated cardiovascular lesion.

DR. PREBLE: And here we have to do with a woman forty-two years of age, suffering from a condition which, whatever its nature, is prolonged, and one would say that maybe the dyspnea present in her case is due not so much to toxemia as to cardiac failure. We have also some elements of toxemia present associated with those of mechanical failure on the part of the heart. So that, whichever way we look at it, the natural conclusion is a question of either the heart or the kidneys, or both, which is more likely. At her time of life it is rarely that we see cardiac disturbance unaccompanied by some kidney affection. In young individuals we may find either one alone, but at this time of life they are usually associated. What complications would you expect?

DOCTOR: Either stasis or toxins from the kidney, or both combined.

DR. PREBLE: Look at the patient.

DOCTOR: She is yellowish.

DR. PREBLE: A little yellow. But what is the first thing that strikes you? She is so dyspneic that she has to sit up in bed. She cannot lie flat at all. That is the most striking thing.

DOCTOR: One notices the blood-vessels on the face.

DR. PREBLE: Yes, the vessels on the cheeks are a little distended.

DOCTOR: The patient coughs a good deal.

DR. PREBLE: Yes. Is she cyanotic? The color of her hands particularly is bad.

DOCTOR: The eyelids are somewhat swollen.

DR. PREBLE: No, not now. They have been. She had perceptible edema about the face, but now the eyelids are scarcely swollen. She has, however, marked edema elsewhere. There is a good deal of edema of the extremities and more or less of the back. When she first came into the hospital the only place where there was edema was over the sacrum and lower portion of the back; also of the face. There was no edema of the feet. Since then the edema of the feet has developed.

As one looks at the patient, the things that strike one are the obvious dyspnea, the exhaustion, and edema. She appears to be clearer mentally today than at any time since coming to the hospital. Ever since her admission her mental state has been abnormal for her, and yet one which is quite frequently associated with this condition. She has been obstreperous, unreasonable, and noisy, and has been a source of a good deal of discomfort to the people about her (the other patients in the ward), not because she wanted to be, but because she could not help it; and very frequently these patients, who are toxic from insufficient excretion from the kidneys, are that way. They often are most unreasonable. Patients who are normally quiet and sensible become very surly and oftentimes much worse than this patient has been. That is a condition for which she is not responsible, but it is a manifestation of her toxemia. However, as I said, she is a little brighter and clearer this morning. Before coming into the hospital she noticed periods of stupor, and these have been more or less frequent since her admission.

What do you think about the edema? It involves both legs equally. It has come on gradually. It is more marked since she came into the hospital.

DOCTOR: More a heart edema.

DR. PREBLE: See that breast, and notice how edematous it is. See that edema of the thorax. But today, at least, there is hardly any demonstrable edema of the face. Can we call this universal edema?

DOCTOR: Nearly universal.

DR. PREBLE: Yes, practically universal. Is it moderate or high in grade?

DOCTOR: High.

DR. PREBLE: Yes; particularly is it intense and marked in the lower levels, for example, the breast. The difference between the upper half of the breast and the lower half is very marked. And yet, while the edema obviously is influenced by gravity, nevertheless it is universal in its distribution. What do you think about the case?

DOCTOR: The edema is not due to the renal condition.

DR. PREBLE: If due purely to some renal cause it would not be so general.

DOCTOR: I think the generalized edema speaks for a combination of heart and kidney.

DR. PREBLE: The renal edemas are much wider in their general distribution—practically always universal. Those influenced greatly by gravity are cardiac. But here we have both; that is, the edema is universal in its distribution, but there is also the influence of gravity. For example, on her hands; the edema on the posterior surface is much more marked than on the anterior. How about it?

DOCTOR: I think that is from the heart.

DR. PREBLE: The distribution of the edema, so far as the body as a whole is concerned, speaks for the kidney, and, so far as the various levels are concerned, speaks for the heart, and the two together speak for both. That is the way in cases of nephritis, where the condition has to a very considerable degree progressed, we find this combination. However, when you look at that edema, particularly in the breast, where the skin is thin, it strikes you more like renal than cardiac edema, because there is so little cyanosis; it is so translucent you feel you could look through it. It is shiny, and always gives the appearance of trans-

lucency, which is not found ordinarily in cardiac lesions. It feels much more like renal edema.

[Here Dr. Preble asked the Intern to examine the heart and mark its outline on the skin.]

During all the time she has been in the hospital she has had an infected gland in the neck, which has no relation to the fundamental process at all, but is an accidental affair. The urine has been abnormal throughout. When she came in, the first examination showed albumin in large amounts without casts; such urine as would suggest, in view of the other findings, an old nephritis. On the next examination it contained albumin, hyaline and granular casts, and so it has run right along, always with albumin, sometimes with casts and sometimes without. The urine has never been measured. We never have been able to get the specimen with any degree of accuracy, every time the bowels moved she would lose some urine. The result of the phthalein tests were as follows: At the end of the first hour we got 20 c.c.; at the end of the second hour, 20 c.c., with faint trace of the dye. The next day we got 30 c.c. the first hour; the second hour, 40 c.c., with traces of the dye. The second day we got 65 c.c. at the end of the first hour, and 60 c.c. at the end of the second hour, without the dye. While the existence of passive congestion lessens the function. the effect ought not to be so striking as that.

In this case the first question that occurred to us when the patient came to the hospital was, is the heart the important thing or the kidney? It was really a matter of opinion, but with such an excretion as that one would say the kidneys, and so, also, the toxic symptoms she has shown. We have, therefore, come to look upon the thing as more renal than cardiac, though possessing the elements of both.

No doubt this woman has had her nephritis for a long time, and then gradually renal and cardiac failure developed, so that we have the combined evidence of cardiac failure with renal failure, and she comes in presenting the combination of circulatory and toxic disturbances which are due to that condition.

This woman is forty-two years of age. She is a highly re-

spectable woman. She has worked hard and faithfully. She is rather inclined to be religious. As to former illnesses, she had chicken-pox, measles, whooping-cough, scarlet fever, and illnesses of that sort when a child, but she has been in good health for years.

DOCTOR: Is it one of the cases that could be put in the idiopathic class?

DR. PREBLE: We don't like that term. It is just one of the large group where no etiologic factor is present. Would you think it likely to be specific? Could it be specific?

DOCTOR: I don't know about that. I think it could.

DR. PREBLE: Well, she is forty-two years old. The thought occurs, can it be specific—primarily a vascular process of specific origin, and a renal condition with changes secondary to it? How about it?

DOCTOR: It could be, but it is not probable.

DR. PREBLE: It is not probable. It is a possibility only. Would it make any difference, so far as the handling of the case was concerned, if it were specific?

DOCTOR: Yes.

DR. PREBLE: Perhaps not now. The changes are probably too far advanced. If specific, it has been going on too long, and the changes in the vessels of the heart and kidney have reached a stage of organization, so that they are stable and cannot be removed. In this case one really would not think of that, but with many patients you would think rather seriously of the likelihood of the whole proposition being on a specific basis, and it might be. However, in this patient, with her habits of living, there is some other cause, the nature of which we do not know. What about the heart?

DOCTOR: It is enlarged.

DR. PREBLE: What else does it show?

DOCTOR: It is regular.

DR. PREBLE: Tones pure or impure?

DOCTOR: I don't know.

DR. PREBLE: Listen again. Her blood-pressure has been running steadily up. She objects to its being taken, however.

It has been 200 systolic and 130 diastolic, 212 and 144, 224 and 130. It has always ranged high, about 200, sometimes as high as 230.

So far as the heart is concerned, she has evidences of cardiac hypertrophy, but it is not due to any valvular defect. The tones are pure. The heart has been rapid, but is not just now, the last count being only 68.

The lungs show a moderate amount of edema and bronchitis, but no consolidation at all.

The abdomen has not shown any fluid.

The edema has gradually increased since coming to the hospital, becoming more marked and severe. The toxic phenomena have grown steadily worse, but there has come a period of remission so far as the toxic condition is concerned. She is less toxic at the present time than when she came under observation. But, as stated before, the edema is worse. So far as one can estimate the excretory power of the kidney with the phthalein test, there is none. So far as the excretion of the fluid is concerned, the amount of fluid excreted is greater than when she first came in, and yet, in spite of that fact, the edema has steadily increased in degree. What would you do for that case?

DOCTOR: She shows some symptoms of broken compensation, and I would put her on digitalis.

DR. PREBLE: In spite of a blood-pressure of 230?

DOCTOR: The elimination is at fault. She might have a blood-vessel rupture.

DR. PREBLE: What are we dealing with? A desperate situation, are we not? And we cannot handle it with kid gloves. We have got to take a chance. Of course, theoretically, anybody would say that with a blood-pressure of 230 or 240 to give digitalis is not a good thing, and yet that is what we did immediately. It did not do her any good.

DOCTOR: You might give her something to bring the pressure down, such as nitrites.

DR. PREBLE: Yes, that would occur naturally, and yet we have not done it because it won't do any good. The elevation of blood-pressure is a conservative process. If we gave nitrites

to lower it, her general condition would immediately get worse. So that won't do at all.

DOCTOR: Why get worse?

DR. PREBLE: Because apparently the minute you lower the blood-pressure with nitrites or anything else which brings about a sudden diminution, the excretory functions are reduced immediately. The intoxication rapidly increases in grade, and one cannot do that very many times before he is convinced that it is not right. To reduce the blood-pressure by reducing the toxic process is the rational thing, but to reduce blood-pressure at the expense of increasing the poison is another thing. What would you do about that?

DOCTOR: You might do a venesection.

DR. PREBLE: With what idea?

DOCTOR: To get the toxins out that won't get out of the system any other way.

DR. PREBLE: How much would you bleed a patient like this one?

DOCTOR: Two pints.

DR. PREBLE: How much did you bleed her? (Speaking to assistant.)

INTERN: A little over a pint.

DR. PREBLE: That is what one should do. When she first came into the hospital the impression she made on me was that the condition was rather more cardiac than renal in nature, and in consequence of that she was put on digitalis, which accomplished nothing except to show us that the condition was rather more renal than cardiac, and as the days have gone by the renal element has become progressively more and more important. The toxic phenomena have become more striking. The circulatory phenomena, while increased in degree, have become relatively less important than they were, less important because of the increasing degree of intoxication, and so we bled her. With what effect do you suppose?

DOCTOR: I imagine that she was better.

DR. PREBLE: If better now, how bad before? As a matter of fact, it did benefit her, and she has been better, but the im-

provement has not been very great. She is not so obstreperous or noisy, a good deal more reasonable, and a little clearer mentally, but the amount of benefit has not been very great. Would you bleed her again?

DOCTOR: No.

DR. PREBLE: Well, I don't know. Of course, that was not a very large section, scarcely over 16 ounces. While not a very large woman, and she has not been well nourished for a long time, still she could perhaps stand another venesection, and it might perhaps be the best thing for her. What other thing could you do?

DOCTOR: Give her a protein-free diet as nearly as possible.

DR. PREBLE: Yes. The real truth of the matter is that we may be grateful for whatever she may eat. Her appetite is very poor. What are the things you want to do in a case of this kind?

DOCTOR: Eliminate the toxins.

DR. PREBLE: Yes. Free the body utterly from toxins if you could. That is, if you knew how. The venesection withdrew a good deal. One can get rid of more toxins in less time by a section than in any other way, but, of course, you cannot bleed them to the point of exsanguination. But you can accomplish more in a minute than in any other way, and that we have done in this case, and I think one might consider the advisability of repeating it. What else could we do in the way of eliminating the toxins?

DOCTOR: Look after the elimination through the bowels.

DR. PREBLE: Yes. In what way?

DOCTOR: Give her a purgative.

DR. PREBLE: Of what sort?

DOCTOR: You might give her elaterin or magnesium sulphate.

DR. PREBLE: Yes, in a general way; however, we prefer the vegetable bodies rather than the salines. Elaterin and compound jalap powder are good. The patient is being given elaterin, $\frac{1}{10}$ grain every hour for four hours in the morning at five, six, seven, and eight o'clock. What is the idea in giving it in that way?

DOCTOR: To get better action. I don't know any other reason.

DR. PREBLE: The idea is that usually they get over the action of it by night, so that their sleep is less disturbed. If you give them the same quantity distributed throughout the day, then they do not sleep so well. Is there any other way to stimulate the excretion?

DOCTOR: I suppose by elimination through the skin. Sweating I don't think would do it.

DR. PREBLE: No. She is too dyspneic. In a patient who has to sit up as she does that would be rather a risky think to do. The amount of elimination you would get in that way would not be very great. You would get rid of a good deal of fluid and a little salt, but not much else; of the solids that we can measure there are none very important. But still oftentimes it is a good thing. Some other patients in the house are being sweated, but with this patient I would rather hesitate to do it. I would much rather bleed her again.

DOCTOR: How long did the blood-pressure stay down after the last section?

DR. PREBLE: You know we must be prepared for disappointment. When you bleed patients of this type you can bleed them a pint or two or sometimes three, and the blood-pressure will go down 10 or 15 mm., that is all. There is not any particular effect on the pressure, but you see the effect in other ways. Her pressure was practically unaffected by the venesection. That is usually the way; the immediate effect on the pressure is not very great, but later on the pressure falls somewhat, but not to a marked degree.

DOCTOR: If it does fall, how long does it stay down?

DR. PREBLE: That depends upon circumstances. There have been a number of other questions to deal with in this case. What would you do for her sleep? She has been unable to sleep.

DOCTOR: To what do you attribute the loss of sleep?

DR. PREBLE: The effect of the toxins.

DOCTOR: Then the other remedies for the toxins would help that.

DR. PREBLE: The things which tend toward elimination tend toward re-establishment of sleep. But they have not done it sufficiently.

DOCTOR: You might give her drugs like veronal, which is one of the best things.

DR. PREBLE: You might give her veronal. What else?

DOCTOR: Chloral, but I don't know what effect chloral would have on her.

DR. PREBLE: Chloral and bromids. How about morphin?

DOCTOR: Yes. You might give her morphin.

DR. PREBLE: Yes; but how about the wisdom of it?

DOCTOR: It has a good effect on the heart.

DR. PREBLE: What effect on the elimination.

DOCTOR: Very poor.

DR. PREBLE: Yes. But sometimes (that is particularly true in a ward patient) you have to do things that perhaps you would not do otherwise. Take this woman, for instance; she would have had every other patient in the ward awake. We had to make her sleep. Chloral and bromids had no effect, and we had to give her morphin, not so much on her account, as on account of her keeping the others awake.

DOCTOR: How often was it given in this case?

DR. PREBLE: One-quarter grain a week ago, and then she kept quieter, so that she did not have to have it again. Night before last she had chloral and bromids, and last night chloral, bromids, and codein.

DOCTOR: Is morphin dangerous in these cases?

DR. PREBLE: I don't think it is as dangerous as reputed. But morphin is excreted very largely through the kidneys, and one has to be careful on account of the danger of accumulation. In heart cases it can be used quite freely, but in renal cases you have to be a little cautious about it. Do not use it if you can avoid it. Chloral and bromids are all right. They would take the place of veronal, as a rule, although if you use them you will occasionally wish you had not.

What is the prognosis here?

DOCTOR: Poor.

DR. PREBLE: What will be the best you can ask?

DOCTOR: That the edema should subside and her dyspnea improve somewhat.

DR. PREBLE: The respiration is the important part of the whole thing. The toxic condition might be lessened and the edema disappear, and that in her might continue for how long?

DOCTOR: For a short time or a number of years.

DR. PREBLE: Not likely a number of years. She might go a few months, but most likely not. What is much more likely to happen is that the condition will grow rapidly and progressively worse unless interrupted by some other critical phenomena that you see in cardiorenal cases, for example?

DOCTOR: Cerebral hemorrhage; cerebral thrombosis; angina pectoris.

DR. PREBLE: Yes. Uremic coma; uremic convulsions; manifestations of the toxic phenomena present in cases of this kind, such as an infarct of the superior mesenteric, and then what would happen? So the best we can hope for in a case of this kind is that a temporary amelioration will take place, which is unlikely, because, in spite of being here for two weeks, she is definitely worse than when she came in, and I think she will continue to get worse. However, you never want to be too sure about these cases. There is nothing in the world that will deceive you more utterly and frequently than the cardiorenal case so far as the prognosis is concerned. With her you will say that everything points toward a rather brief clinical course, and yet she may pull out. No matter how desperate they are, they occasionally pull out. You would say that a woman in the condition she is in, toxic and edematous, with such poor renal excretion, would certainly and inevitably go to pieces within the course of a few days, but she may not.

CLINIC OF DR. MAURICE L. GOODKIND

MICHAEL REESE HOSPITAL

PNEUMONIA. PROGNOSIS ACCORDING TO THE GIBSON RULE

TODAY we shall begin the study of an acute infection which occurs with great frequency at this time of year. I have selected two patients for demonstration: One is sixty years of age and the other seventy. Both began with a pronounced chill, but in all other respects they differ very radically in their symptomatology.

The first patient, Mr. I. S., seventy years of age, has always been in a fair state of health until a week ago. At that time, without any premonitory symptoms, he had a very decided rigor. He then developed a violent delirium suggestive of an alcoholic mania, although he has never been addicted to alcohol; very severe headache; some rigidity at the nape of the neck; temperature at the time of admission to the hospital was 103° F. per rectum, pulse 110, respirations 24; Kernig, Brudzinsky, Gordon, Oppenheim, and Babinski are all negative; ocular movements and reflexes are normal. Urine contained some albumin and few hyaline casts.

I wish to emphasize the fact that at no time did this man have any symptoms referable to a disturbance in his lungs: no cough, dyspnea, expectoration, herpes, or pain in the chest. But as we proceed to examine him we find dulness extending over the entire right lung, anteriorly and posteriorly; loud bronchial breathing is also heard over this area, bronchophony is elicited; heart sounds are clear and distinct. Blood-pressure: systolic, 160, diastolic 100, pulse-pressure 60; blood-culture is completely negative after four days' observation. The white blood-count is 16,000, with 85 per cent. polynuclear leukocytes.

We have an acute lobar pneumonia in an aged man with all the physical signs well marked, giving none of the ordinary symptoms of the disease.

This class of cases in the very aged and the very young simulate cerebrospinal meningitis or acute abdominal conditions (such as gall-bladder and appendicular inflammation or intestinal obstruction), and may lead one into serious error unless a very careful physical examination is made.

On examination of our next patient we find the antithesis of the symptomatology of the former. Here we have a pronounced cough; dyspnea as evidenced by the rapid shallow breathing and forcible movements of the alæ nasi; reddish flush of the left cheek; constant restlessness; short cough associated with expectoration of glairy, viscid, blood-stained sputum; severe pain in left chest.

As we percuss we find a dull tympany over isolated areas of the left lung with bronchovesicular breathing; pulse of 130, with a blood-pressure of 90, which signifies, when interpreted according to Gibson, that *when the systolic pressure curve, expressed in millimeters of mercury, is lower than the rapidity of the pulse curve, representing frequency per minute, an unfavorable result may be apprehended.*

In the treatment of pneumonia we have run the entire gamut of therapeutic agents—viz.: excessively large doses of digitalis and of potassium iodid, huge subcutaneous injections of quinin and urea, serums, and vaccines, without in any way appreciably convincing ourselves of their utility.

We shall keep these patients in cool, well-ventilated rooms, and pay adequate attention to the diet so as not to increase their burdens through the addition of an intestinal toxemia.

In case of the sthenic patient with a strong pulse and vigorous heart action an ice-bag may be used for temperature, together with appropriate hydrotherapy. While in the case of the patient with low blood-pressure attention shall be directed to sustaining the heart action and the vasomotor tone with epinephrin, strychnin, diuretin, and whisky.

We have had several collapses on the part of this patient in

which we have been compelled to administer strophanthin intravenously, together with hypodermic injections of camphor. On one occasion the engorgement of his venous system was so extreme that venesection was performed, which brought him relief.

In this asthenic type of patient cloths wrung out of warm water to which mustard has been added, and applied to the chest, served the purpose better than the use of cold.

A further indication of the lowered vitality and lessened resistance of this patient is the low white blood-count.

[Note.—Five days later: The patient who seemed to be progressing favorably in all respects except that his systolic blood-pressure remained low, had a return of auricular fibrillation, and, despite heroic measures which had been of service in previous attacks, fulfilled the expectations of the Gibson rule and the case terminated in an exitus within two hours.]

TABES. HYPOSENSITIVE CONDITION OF THE ANUS AND URETHRA IN THE INCIPIENT STAGE. SWIFT-ELLIS TREATMENT

The next group of patients which we shall proceed to study are three men in the fourth decade of life, all with preceding histories of Spirochæta pallida infections. Paraplegia, optic atrophy, visceral crises, lancinating pains, the Romberg, Westphal, and Argyll Robertson pupil characterize all, so that no difficulty should be experienced in arriving at a correct diagnosis in this advanced type.

It is in the early stages of the disease that you must be on the alert in order to diagnose correctly, for then much can be done to arrest its rapid development. Through failure to make a systematic examination many cases in the incipient stage are erroneously called myalgia, neuritis, rheumatism, biliousness, neurasthenia, and other mistaken names. Among the tests of

good collateral value in these early cases, and which in my opinion rank in importance with the ulnar and testicular insensitiveness, are the signs described by Collier Martin and myself. Collier Martin has shown that energetic manipulation of the anus causes very little discomfort, and that light lateral pressure causes rapid relaxation of the anal muscle. For many years I have called attention to the marked insensitiveness of the urethra, on account of which it frequently happens that a patient urinating in the dark is unable to appreciate the fact, and, again, manipulation with sounds in the urethra fails to produce the usual pain. Signs of this kind in conjunction with observed symptoms are often suggestive of the presence of an early tabes.

In two of these patients the blood-serum gives a marked Wassermann reaction, with a very slight response in the third, but in all three the spinal fluid presents a very strongly positive Wassermann. We continue to use the Lange colloidal gold test, but find the results so conflicting that we regard it as of but slight value.

In all there was a very marked globulin reaction, as determined by the Pandy, Nonne, Ross-Jones, and Noguchi tests, and a marked pleocytosis of the lymphocytic type.

But this has been very materially changed through the subdural method of treatment advocated by Swift-Ellis, which we now practice in our advanced and intractable cases, with some modifications from the original method. One of the departures is the withdrawal of the serum within forty minutes after the intravenous injection of neosalvarsan, as the Abelin procedure for the detection of even the most minute trace of arsenic has demonstrated the presence of arsenic in the serum within twelve minutes after the intravenous introduction of salvarsan, and its absence in from sixty to ninety minutes. Second, we add our salvarsanized serum to a hypertonic salt solution instead of a normal salt solution as being less irritating. Third, $\frac{1}{50}$ grain of mercuric bichlorid is added to the hypertonic salt solution before introducing it subdurally.

In all we have had about 20 cases to whom we have admin-

istered this form of treatment, and, although we have not had the brilliant results reported by some authors, in the majority of our patients some very definite improvement has been noted, such as the sterilization of the spinal fluid, which had failed to respond to the most vigorous use of arsenic and mercury otherwise administered. Also the pains have been very greatly ameliorated, visceral crises abated, and a general improvement in health obtained, so that we feel encouraged to continue its use in appropriately selected cases.

CHOLELITHIASIS. DIAGNOSIS BY EXCLUSION

Referred for Operation to Dr. E. Wyllys Andrews

The patient presented in this clinic for consideration today is a married woman, thirty-three years of age. She has had six children, no miscarriages. History of past illnesses and infections, such as typhoid fever, is completely negative. Family history is negative. She enters the hospital complaining of severe pain in the epigastrium which comes on within a short time after partaking of food. The pain is of a sharp, stabbing character, beginning in the epigastrium and radiating to the left side of the chest wall posteriorly toward the scapula; it is never reflected to the right shoulder, to the right mamma, or beneath the right costal margin; it is occasionally associated with spells of vomiting which brings up the ingested food. Never at any time has any blood been observed in the vomitus; this emesis does not appear to relieve the pain, nor is it relieved by heat, change of position, or alkalis; it is never accompanied by chill, fever, or icterus. Occasionally acid eructations are present, which also fail to bring relief. These symptoms have persisted over a period of several years, with marked exacerbation during the past six months. Her intestinal function is

sluggish, constantly requiring artificial aid. The stools at no time have been acholic or tarry. Since the advent of her trouble she has lost about 30 pounds.

Physical examination reveals a somewhat emaciated appearing woman, otherwise there is nothing significant in her general appearance. We find no icterus or cachexia. She has a pulse frequency of 80, systolic pressure of 120, and diastolic of 90 on the right side; on the left side systolic pressure of 100, diastolic of 80. Here I wish to emphasize the frequency with which discrepancy is found in the pulse-pressure of the two sides, the greater pressure being usually observed on the right side. Her temperature was 98.6° F. on admission to the hospital three days ago, and has shown no departure from the normal since then. Lungs and heart perfectly normal.

On examination of the abdomen we see a prominence underneath the right costal margin which moves to and fro with respiration. On percussion it appears continuous with the liver dulness. On palpation it is painful, is kidney shaped, and moves freely with respiration. It is of semi-elastic consistency. Beneath this mass, and apparently connected with it, one can palpate another mass about the size of an orange which can be shoved backward and downward, and which disappears on inflation of the intestine. It appears to move in conjunction with any movement on the part of the upper mass, and cannot be separated from it by any manipulation. The patient has no ascites, enlarged veins, or adenopathy.

The urine is acid, contains no albumin, but gives a slight reduction test which is found to be lactose (the woman recently weaned her nine months old baby). There were no casts; bile has been present only on one occasion. The urobilinogen test (dimethylparamidobenzaldehyd 2 per cent. in 5 per cent. HCl) was strongly positive.

The blood examination gave hemoglobin of 72 per cent., red blood-cells 4,640,000, white blood-cells 7400, of which 72 per cent. were neutrophiles.

The gastric analysis, made forty-five minutes after an Ewald meal, yielded 20 c.c. of well-digested mass, with total acidity

Fig. 23.—Excised gall-bladder, showing large size.

Fig. 24.—Interior of excised gall-bladder, showing stones.

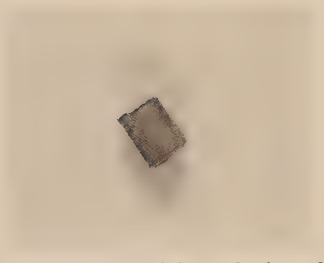

Fig. 25.—Photograph of a rivet removed from the bronchus of a child. Actual size.

of 90, and free of 60, no organic acid, no blood; motor meal was negative.

The stools contained no blood and there was no evidence of disturbed pancreatic function; bile was present.

Fluoroscopic examination reveals a normal contour of the stomach; there was no retention of the bismuth-buttermilk meal beyond the normal limit; the pyloric cap was well formed. No Haudek spots or distortion of the wall. The bismuth column was not delayed in its passage through the intestine; the head of the column appeared at the splenic flexure in about eighteen hours.

The Röntgen plates showed an indefinite shadow in the hepatic and right renal region.

The cystoscopic examination was negative.

To summarize briefly: We have a female patient thirty-three years of age, with a history of dyspeptic symptoms extending over a period of seven years, marked increase of the symptoms during the past six months. The laboratory findings other than a hyperchlorhydria were negative.

The physical signs were those of a mass lying underneath the liver, evidently connected with it and the right kidney.

From the normal blood-picture—absence of eosinophilia, leucocytosis, or anemia—the absence of chills and fever, of palpable thrill and nodules, antecedent history, we are justified in excluding abscess, carcinoma, and echinococcus of the liver.

In our judgment the differential diagnosis rests between involvement of the gall-bladder or cyst of the right kidney, or a combination of both. And in this connection I wish to cite three cases that recently came under my observation in which the kidney element was entirely overlooked: one was an involvement of the lung; the second, of the intestine; the third, of the pericardium. On postmortem these were found to be metastatic masses from a hypernephroma which at no time produced any renal or adrenal symptoms, such as pigmentation of the skin or changes in the blood-pressure, albumin, or casts, so that the kidney and adrenal origin of these cases was entirely overlooked.

I also wish briefly to cite a case in which a very similar mass was found in the same location and of the same consistency as that of our present patient, and which originated in a ruptured duodenal ulcer with a walled-off inflammatory exudate. This condition, as well as the appendicular origin of this trouble, can be excluded.

We shall call upon our colleague, Dr. E. Wyllys Andrews, to perform an exploratory laparotomy, so that we may definitely decide the nature of this woman's trouble.

Subsequent Report

At the operation a huge gall-bladder, about 10 cm. long and 5 cm. wide, with thick congested walls, was found and excised. The right kidney, enlarged and movable, was attached to the under surface of this mass. On opening the gall-bladder it was found to contain one large stone about the size of a bird's egg and innumerable smaller ones.

The patient is now making an uneventful recovery.

FOREIGN BODY IN THE BRONCHUS SIX MONTHS, WITH COMPLETE RECOVERY AFTER REMOVAL

Our next patient is little Charlie L., five years of age, brought to Michael Reese Hospital through the courtesy of Dr. Yates, of Bonfield. This little lad had been well up to the onset of his present trouble, which began last October. Since that time he has lost considerably in weight, has daily chills and fever and night-sweats, with a harassing, stridulous, non-productive cough. At the time of his entrance to the hospital the child's temperature was 104.8° F., pulse 148, respiration 34.

Physical examination shows a decreased mobility of the right thoracic arch, dulness over the entire right side of the chest anteriorly and posteriorly, diminished repiratory note, and

absence of breath sounds over this area. There was no Grocco. He had a leukocyte count of 38,000, of which 85 per cent. were polyneutrophiles. This child at various times has been treated for malaria and tuberculosis until he came under the charge of Dr. Yates, who, in closely interrogating the parents, determined that this array of symptoms came on shortly after the child had had a rivet in his mouth. The doctor assumed that this possibly might have been aspirated into his lung. This assumption was easily verified by the Röntgenoscopic examination. You see at the juncture of the trachea with the right bronchus a foreign body. We have asked our colleagues, Dr. E. Fletcher Ingalls and Dr. Stanton A. Friedberg, who are especially adept in removing foreign bodies by the bronchial route, to attempt a bronchoscopic removal.

APRIL 14, 1915.

I am glad to be able to report to you the successful extraction of this foreign body by Dr. Ingalls and Dr. Friedberg. An initial attempt of fifty minutes was futile, but was followed three days later by the successful extraction of the rivet within ten minutes before the Röntgen screen; it is about the size of a 44-caliber cartridge.

Since the extraction of this foreign body the child has lost its cough and temperature and all untoward symptoms have completely disappeared. The physical signs are also gradually clearing up.

We can regard this as a most fortunate issue, for, in the majority of cases, after a foreign body has been lodged for so long a period within the air passages it is prone to leave behind, even after successful removal, an abscess, gangrene, tubercular process, or bronchiectasis, which may remain unilateral or also involve the opposite side.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

SYPHILITIC AORTITIS; A DIFFERENTIAL DIAGNOSIS OF LESIONS OF THE AORTIC VALVE

THE first patient to whom I direct your attention entered the hospital on the 31st of last December. The history which was obtained at that time and recorded here is to this effect: The patient says that up to two years ago she was in excellent health. At that time she was seized suddenly with attacks of fainting; became unconscious. When consciousness returned she was very dyspneic, and since that time she has had recurring attacks of shortness of breath and edema of the feet. She has also had attacks of palpitation, accompanied by cough, with pain in the upper part of her abdomen.

She has been in various hospitals several times with these recurring attacks. There is usually a gradual improvement, with subsidence within a month. For the past two years her dyspnea was particularly marked upon exertion, and there has been swelling of her feet and palpitation of the heart and a considerable amount of disturbance on the slightest exertion.

Her present attack, as given here, appeared about the 1st of December; then for about two weeks she was comfortable or fairly comfortable, and was able to do ordinary housework.

Just before she entered the hospital, on the 15th of December, as already related, she became definitely dyspneic, with pain in the region of the heart and epigastrium, and the appearance of edema.

On the morning of the 31st, after ascending two flights of stairs, she became very dyspneic and cyanotic. Her condition was so alarming that a doctor was called and she was at once sent to the hospital.

Past History.—She says she suffered from scarlatina when a child; a diagnosis of pleurisy was made in November, 1914.

Family History.—Her father is living and well; mother died of appendicitis; she has two sisters and one brother, all living and well.

She was married; her husband died some years ago, as recorded here, by suicide.

There is no history that this patient has ever been pregnant.

Venereal and Personal History.—She admits a luetic infection ten years ago which was contracted from her husband. The primary lesion was on the labia followed with the usual secondaries.

There is a history of a moderate use of beer, but this only in moderation.

The examination of this patient when she entered the hospital was practically the same as it is today. The chart reveals a pulse-rate of 120 on admission. This continued 120, 124, 126 for the first four days, when the pulse-rate fell to 80, and has continued somewhere in the neighborhood of 80 since that time. Her temperature, as recorded on many occasions, has reached $99\frac{1}{2}^\circ$ or 100° F., and this temperature usually occurs in the afternoon. On other days the temperature remains normal—98.6° F. The respiration rate is 20, 22, and 24, with no marked increase during the attacks of dyspnea.

We will next direct our attention to some of the physical findings. The patient, as you will observe, is in a semiprone position, made necessary because of her dyspnea; when she lies flat, in the dorsal position, her dyspnea becomes more pronounced and she suffers from attacks of pain. The pain, as a rule, is definite and circumscribed in the region of the heart, or the pain may become more intense over the base of the heart, and is quite likely to be referred to the left shoulder and down into the arm. These attacks of pain occur at irregular intervals. She may go two, three, or four days without pain, or, on the other hand, she may have two, three, or four severe attacks during twenty-four hours. Yesterday, in the afternoon, she had one quite severe attack, but the pain was chiefly limited to the

region of the heart and not referred to the shoulder or into the arm. On Friday of this past week the pain was most intense in the shoulder and in the arm, and not so severe in the region of the heart.

Aside from the position of this patient, we also observe no definite cyanosis either of the lips, the ears, or the finger-tips. There is no jaundice, no abnormal pigmentation of any kind, but our attention is arrested, in our inspection of the face, by the presence of the very definite whitish sclerotic scar involving the right half of the upper lip and the right side of the nose. We also observe the presence of an irregular, whitish, sclerotic area over the right temple, accompanied by a definite increase of the pigment about the border.

When this patient entered the hospital in December both of these areas were ulcerated and discharging. Since that time they have both undergone healing, and at this time present only scar-formation.

As this patient refers the majority of her symptoms to her chest, and particularly her heart, we will direct our examination first of all to the heart.

On inspection we find an increase in the heart rate as indicated by the cardiac impulse. We will also observe the location of the apex-beat outside of the nipple line, midway between the nipple and axillary line, rather circumscribed, and located in the sixth intercostal space.

On palpation we confirm the location of the apex-beat, also observing that the impulse is rather strong, forcible, not particularly increased in area above the normal, accompanied by a very short thrill before the apex-beat; in other words, a presystolic thrill. Over the base of the heart no thrill, no definite cardiac impulse. We also notice upon light pressure over the region of the precordia there is definite tenderness. This is a condition of which the patient complains particularly after one of the attacks of pain either in the region of the heart or when pain is referred into the arm; it is more definite over or near the location of the apex-beat, not over the base of the heart. No tenderness or pain in the arm or the forearm.

On percussion we find hepatic dulness at the fifth right intercostal space. The right border of the heart is over a full finger's breadth to the right of the sternum. The base of the heart is at the second intercostal space. The left border is practically at the anterior axillary line. Over the base of the heart and aortic area there is definite increase of percussion dulness a full finger's breadth to the left.

On auscultation at the apex the two sounds are present. Before the first sound a definite presystolic murmur or rumble, running into the first sound and continuous with a systolic murmur; the first sound is not accentuated. With the second sound there is also a definite murmur. Over the fourth intercostal space, to the left near the sternum, we hear a systolic and a diastolic murmur. Over the second intercostal space to the left, or the pulmonary area, there is heard a systolic and diastolic murmur, but more intense than over the fourth intercostal space. Over the second right intercostal space as well as over the first intercostal two tones can be heard—a very loud systolic with a fainter diastolic. Over the tricuspid can be heard a faint systolic and fainter diastolic murmur.

I have already noted the rate of this pulse—previously increased, this morning at 96; now relatively increased, probably from temporary excitement. The pulse rather decreased in volume, and not giving you the impression of a quick, collapsing character.

The tracings made from this patient confirm the impression which we obtain on palpation. We observe, first of all, an irregularity in these tracings. These pulse-beats are not recorded at a regular period; here we have three, possibly four, at regular intervals, and then one which has occurred before the regular time. You will observe, after an increase in the period, that one, two, three, four, five, or six beats occur at fairly regular intervals; then a pause or a prolongation; and the same thing has occurred later with four hurried beats, and then again a return to a fairly regular rhythm. The same is also true in the second tracing.

Examination of the abdomen: There are no abnormal findings on inspection or palpation; no rigidity, no tenderness through-

out the entire abdomen; the spleen is not palpable; the liver is not palpable.

On percussion the lower edge of the liver is at the costal margin. Normal tympany over the entire abdomen.

The examination of the lungs is also negative.

On careful examination, at least during the time when she is free from pain and dyspnea, there are no pulmonary findings. At the time of the attack of dyspnea, and particularly of the pain, râles are usually present over the entire chest, anteriorly and posteriorly.

The pupils are equal, respond promptly to light and to accommodation.

Reflexes: Patellar slightly increased; no ankle-clonus and no Babinski.

DOCTOR (addressing Patient, referring to sore on knee): When did this sore appear?

PATIENT: It came of its own accord; it came a long time ago.

DOCTOR: When did it heal?

PATIENT: A long time ago.

DOCTOR: On the inner side of the right knee there is a small slightly depressed scar, irregular in outline, rough, elliptical, with definite increase of pigment about the border. This sore antedated the sores in the temporal region and on the side of the nose, and its healing antedated the appearance of the lesions on the nose and face.

DOCTOR: This was entirely healed before the others appeared?

PATIENT: Yes.

DOCTOR: Now we have the history of this patient and the physical findings as indicated; what is the diagnosis?

INTERN: There is mitral stenosis; mitral insufficiency; aortic insufficiency; also an angina.

DOCTOR: We direct our attention, as we have in our examination so far as diagnosis is concerned, first, to the heart. We can make this diagnosis, that the patient has an endocardial involvement. She has a cardiac involvement presenting clinically the signs and symptoms of endocarditis.

On the basis of the displaced apex-beat downward and outward, from the increase in the cardiac dulness, particularly in the transverse diameter, we can make the diagnosis, in the presence of the diastolic murmur heard over the base of the heart, of an aortic incompetency. A diastolic murmur is to be heard in the aortic area as well as the tricuspid area and at the apex of this heart. At the mitral and tricuspid areas the murmur is of the same quality as the diastolic murmur heard over the aorta. The maximum point of intensity is at or near the aortic area, and we feel justified in saying that the murmurs heard over the tricuspid and mitral areas are transmitted from the aortic area.

Then there is at the apex the rumbling, presystolic-systolic murmur, the presystolic occurring and localized at the apex, a crescendo murmur, unaccompanied by accentuation of the first sound, but accompanied by a presystolic thrill.

Remember also the character of this pulse. That the pulse is rather decreased in volume, not full and bounding, and particularly not of the collapsing character found in aortic incompetency. The systolic murmur at the apex, transmitted to the left, indicates a mitral insufficiency.

The endocardial diagnosis, then, would consist of an aortic insufficiency and a double mitral, an insufficiency and stenosis.

The significance of the systolic murmur heard at or near the aortic area and the murmur of maximum intensity heard over this area brings up the consideration of several different factors. We have already indicated the absence of a thrill over the base of the heart. A very definite pulsation is present in the supraclavicular area, but no thrill. There is no definite abnormal pulsation in the intercostal spaces to the right or left over the base of the heart, but on auscultation there is a loud systolic murmur.

Doctor: What are some of the conditions with which this murmur may be associated? What are the causes of a systolic murmur over the base of the heart or over the aortic area?

Intern: Possibly a transmitted systolic murmur.

Doctor: Yes, but what?

INTERN: A systolic mitral or tricuspid, or a pulmonary stenosis.

DOCTOR: What are some other causes?

INTERN: Aortic aneurysm, atheroma of the aorta, dilatation of the aorta.

DOCTOR: What are some other causes?

INTERN: Aortic stenosis or syphilitic aortitis.

DOCTOR: Yes. Aortic stenosis. Probably one of the most frequent errors made in physical diagnosis is to interpret the presence of a systolic aortic murmur as indicating an aortic stenosis. As a matter of fact, it is comparatively rare, but a systolic murmur at the base of the heart is frequent.

Now, in order to arrive at a diagnosis, we will indicate the differential points for our consideration.

First, as to the matter of an aortic stenosis: The three cardinal signs in the clinical recognition of aortic stenosis consist, first, of the systolic thrill over the base of the heart; second, systolic murmur in the aortic area, transmitted upward in the direction of the current of the blood; and, third, interference with the pulse; that is, the pulse is decreased in volume, slightly slower, gradual filling, and collapse.

We have here two of the three cardinal signs: Over the base of the heart a systolic murmur transmitted upward and an interference with the pulse. We can, however, disregard the character of this pulse for the reason that we can account for it on the basis of interference at the mitral area; consequently, we have left one of the three cardinal signs, the systolic murmur over the base of the heart. It is the very occurrence of a systolic murmur on auscultation that usually leads to a diagnosis of aortic stenosis—and an error.

Another condition, which is comparatively frequent and far more frequently the cause of a systolic murmur over the base of the heart, occurring after middle life, is when the vessels become slightly sclerotic and the aorta dilated from atheroma. This, however, can usually be readily differentiated from aortic stenosis by one factor, and that is the character of the second aortic sound. If the second aortic sound is present and increased

in intensity, accompanying the systolic murmur, the findings indicate a dilatation.

In aortic stenosis the second aortic sound is not increased in intensity and is quite frequently entirely absent, due to the involvement of the aortic leaflets, and not infrequently there is an accompanying diastolic murmur of aortic insufficiency.

As to the occurrence here of aneurysm: It is true we have the history of pain in the region of the heart, radiating down into the left arm, a common manifestation of an aneurysm, but we have no physical signs upon which to base the diagnosis of an aneurysm. No difference in the radials; no tracheal tug; no pressure symptoms or signs, and we would have to exclude quite definitely the existence of aneurysm.

There is, however, another condition which was mentioned and of the greatest importance, and that is the matter of a syphilitic or luetic aortitis. This is a condition that we must consider here very seriously, not only because of the history (the admitted infection of ten years ago), but also because we have other physical signs strongly indicating the possibility of an aortic involvement. We have here clinical evidence of a cutaneous syphilis. We also have evidence, so far as the heart is concerned, of an endocardial involvement, with the strong probability that the endocarditis is also of luetic origin.

Let us inquire for a few moments into the matter of syphilitic aortitis. This is a condition which was described anatomically years ago by Morgagni. The condition, however, received no attention practically until 1888, when the matter was taken up by Döhle. His report, however, created no particular interest; it only aroused unfavorable criticism, and the condition was practically unnoticed until 1903, when it was again described, and in greater detail, by Chiari, of Prague. Since that time the condition has been very definitely and extensively studied.

From a clinical standpoint, syphilitic aortitis manifests itself in one of four ways: First, by the occurrence of attacks of angina; second, physical signs and symptoms of a dilatation of the aorta; third, by the production of aortic aneurysm; fourth, the production of aortic valvular lesions.

One or a combination of these four is the usual clinical manifestation of syphilitic aortitis.

We have here the history of attacks of angina—definite and typical attacks. We also demonstrated the existence of endocarditis, aortic incompetency with a double mitral. What evidence have we of an aortic involvement? From our physical examination a slight increase in the aortic area and the presence of a definite systolic murmur heard over the same area, the murmur transmitted upward.

We also have, as is the rule in syphilitic aortitis, at least three very suggestive symptoms in our clinical history. These consist of recurring attacks of dyspnea. They suffer from definite attacks of dyspnea, sometimes coming on without any exciting cause, or in other instances precipitated by slight exertion or a little nervousness or excitement. The characteristic type of the dyspnea associated with syphilitic aortitis is that of difficult expiratory breathing. The difficulty occurs more with expiration than with inspiration. This has been described as the pulmonary type of angina, ordinarily referred to, particularly by the Germans, as Lufthunger ("air hunger").

Then the second symptom is that of definite occurrence of attacks of angina. Either the pain is circumscribed, located in the region of the heart, or the more typical attacks referred to the left shoulder and arm.

The patient here has at times suffered from both types of pain due to angina.

And then the third symptom, always suggestive, is the occurrence of a slight temperature. Popoff called attention to the occurrence of temperature and obscure pyrexia, and from his anatomic study attributed the condition to the syphilitic aortitis. Some of these patients will present a pyrexia of 101°, 102°, or 103° F. No other factor can be determined or demonstrated as the cause of the pyrexia, and on specific treatment the temperature disappears. This patient has had 99° or 100° F., and on a few occasions a fraction above 100° F. We believe that we are justified in attributing this pyrexia to the presence of a probable syphilitic aortitis.

The possibility of an accompanying or accidentally associated infection producing acute endocarditis must be borne in mind. A tonsillitis or any focal infection with an acute endocarditis will acount for the temperature. This may be determined by blood-cultures, the enlargement of the spleen, and the frequent occurrence of emboli.

The further diagnosis of a syphilitic aortitis will usually rest upon two points—the presence or absence of a Wassermann and the fluoroscopic or skiagraphic examination.

This patient has had up to the present time two blood Wassermann tests. The first examination was on the 1st of February, and the second examination was on the 8th of February. The former giving a three plus and the second one a four plus. So we have a definite, positive Wassermann.

This simply is confirmatory: We must remember that we have here the admitted history of a primary lesion ten years ago with definite physical signs of cutaneous tertiary manifestations, and we must also remember the endocardial involvement. The Wassermann is confirmatory, and makes the presumption even more likely that there is a syphilitic involvement of the aorta.

Then, as to the examination with the fluoroscope or skiagraph, the condition varies. We usually encounter at least three different types of so-called syphilitic aortitis from the standpoint of the skiagraph. Probably the most frequent picture in the skiagram consists of an elimination of the so-called aortic knob. In the normal, as the aorta turns to the left and downward, there is a little localized bulging, which is designated as the aortic knob. In dilatation, or in syphilitic aortitis with dilatation of the aorta, this knob becomes obliterated, or, because of the filling of the space between the knob and the heart, the shadow becomes continuous, as we have here—the knob has disappeared.

Then, second, an increase of the aortic shadow to the right, particularly where the endocardium is not involved and there is no increase in the cardiac shadow. The aortic shadow extends further to the right than the right border of the heart, and when that occurs it indicates a dilatation of the arch of the aorta. Here that condition is not present, for the reason that we have

Fig. 26.—Large heart shadow with diffuse dilatation of aorta.

this great, enormous increase in the cardiac shadow from endocardial involvement, and the right border of the aorta is well within the limit of the right border of the heart.

Then, third, frequently syphilitic aortitis consists in uniform dilatation of the shadow; that is, there is a definite increase to the right and the left of the aortic shadow. In this picture, however, we have the usual change, which consists in obliteration of the aortic knob. (Shows skiagrams.)

So, then, returning to the matter of diagnosis, aside from the endocardial involvement, consisting of aortic insufficiency and the double mitral lesion, we can establish without any doubt the existence here, on the basis of the history and cutaneous manifestations, the symptoms of angina, attacks of dyspnea, the slight temperature, the presence of a positive Wassermann on two occasions, and the skiagraphic examination characteristic of a dilatation of the aorta, the diagnosis of luetic or syphilitic aortitis.

Prognosis.—Under ordinary conditions, and particularly where the endocardium is not involved, the prognosis is not wholly unfavorable, and particularly with antisyphilitic treatment. These patients with syphilitic aortitis do very well on the salvarsan or neosalvarsan treatment. At first the tendency was to consider salvarsan or neosalvarsan as contraindicated, but at the present time the treatment is employed, and usually with decided benefit. It is our custom to combine with the salvarsan or neosalvarsan treatment also the use of mercury, preferably in the form of inunction, and also mercury by mouth.

This patient when first under observation was confined to her bed, but during the last two weeks she is up and about excepting at the time of her attacks of dyspnea or angina. Ordinarily she is comfortable, and you would hardly arrive at the conclusion from her general appearance and from her activity in the ward that she was suffering from an endocarditis and syphilitic aortitis with an angina.

HOUR-GLASS STOMACH. SOME OF THE DIFFICULTIES ENCOUNTERED IN THE DIAGNOSIS. DISCUSSION OF ACUTE ABDOMINAL PAIN.

THIS patient entered the hospital on the 6th of March. She is twenty-five years old. When she was admitted to the ward she complained of pain in the left side of the abdomen, with shortness of breath and attacks of vomiting. The patient had been well up to about one year ago, when she began to have moderate pain in the small of the back on the left side. At times it became quite severe, and is described by her as being colicky in character. It lasted, on the average, about one day and then gradually subsided. No history of chills.

About six months ago for the first time there was some discomfort when she urinated; the frequency was slightly increased. After a month or two night-frequency increased to four or five times, and there was also increased frequency during the day. At the time of voiding the urine she complained of pain in the left side of the abdomen. On the day she entered she had a very severe attack of pain with sudden onset which occurred early in the morning, and this attack lasted throughout the entire day. She came to the hospital with the pain still of moderate degree.

The urine, as described by her, has often been dark colored, and this was noticed particularly on the first of this month and also for the twenty-four hours preceding the present attack. More particularly in reference to the pain: She has never been completely free from it; according to this history it became worse at times, with the recurring attacks definitely colicky in character, most marked on the left side of the abdomen.

There is a history of loss of weight during the last year, of approximately 15 pounds. She has vomited at frequent intervals, usually accompanying the attacks of pain or colic. She gives a history of having had an attack of gastric disturbance, which was diagnosed as gastritis, two years ago. Three years ago the patient was operated for some abdominal condition. Her family history is negative; no tuberculosis and no cancer in

the family. Personal and menstrual history negative. She has never been pregnant; has never used alcoholics, and denies a venereal infection.

This patient was sent into the hospital with a diagnosis of kidney-stone. The history, as we have it, is concerned with the matter of some abdominal distress and disturbance, associated particularly with the presence of pain, with recurring attacks of a colicky nature, accompanied with nausea and vomiting, with the history of some urinary disturbance; that is, the presence, as described by the patient, of a dark, smoky urine (possibly blood in the urine), and with the pain more definite and more marked in the region of her left kidney.

We have here, then, many possibilities, if we are to consider the condition simply from the standpoint of our clinical history. It opens up the field for the consideration of all of the disturbances associated with pain in the abdomen, and at the outset it becomes necessary to make a distinction between the character and the occurrence of the pain. Whether the pain is of a dull, continuous character, or whether it occurs in paroxysms, coming on suddenly, severe, and probably terminating rather abruptly. The patient states that she is practically never free from abdominal discomfort, but with definite paroxysmal attacks. In the ward she had has many of these attacks, and apparently the pain is intense. The patient often assumes an upright position, complains bitterly of the pain, and it is frequently accompanied by nausea and vomiting.

Among the considerations is that of a kidney-stone, already mentioned. There are other conditions associated with the kidney—the possibility of a movable or floating kidney with a Deitl's crisis; the possibility of some other variety of a colic associated with a gall-bladder involvement, a cholecystitis or cholelithiasis; the possibility of pancreatic involvement, a pancreatitis, a pancreatic hemorrhage, or a carcinoma or neoplasm of the pancreas; the pain and the colicky attacks associated with gastric involvement, acute gastritis, for instance, accompanied with severe pain (remembering the patient gives a history of having had a so-called gastritis a couple of years ago); pain

associated with a peptic ulcer either gastric or duodenal, or the pain due to a carcinoma of the stomach; pain associated with some other condition in the abdomen, perhaps one of the most frequent to be thought of is the existence of appendicitis, and in females the involvement of the pelvic organs, as a salpingitis.

Then the involvement of the intestines, characteristically associated with paroxysmal attacks of pain, in such conditions as intestinal obstructions due to impaction, volvulus, adhesions, or strangulation in the various forms of hernia; or another condition which is by no means infrequent, and one to which we will return later, a mucous colitis. Rectal or colon involvement in the form of hemorrhoids, stricture, or carcinoma are not infrequent sources or causes of attacks of pain of the colicky or paroxysmal type.

Then we have, aside from these considerations, the possibility of a peritonitis, either acute or chronic, or adhesions of the peritoneum, which are responsible for the pain, owing to the effect on the other organs. Another consideration here is the matter of a vascular involvement; the presence, for instance, of an aneurysm of the aorta, the existence of an aortitis, or an abdominal angina.

Then, as far as the nervous system is concerned, the presence of a tabes with abdominal crises,—recurrent abdominal pain with vomiting,—that must always be borne in mind. Also the possibility of a plumbism with the severe paroxysmal attacks of pain; not infrequent and not to be forgotten.

When we turn to the consideration of the case from the standpoint of examination, we find that this patient has a pulse-rate varying from 80 to 98; her respirations remain almost constantly 20 to 23; while the temperature at no time has been above 99° F., almost constantly 98.6° F.

The physical examination of this patient, particularly in reference to the abdomen, on inspection is negative, aside from the presence of a discoloration due to the local application of heat, and also the presence of an old abdominal scar from her laparotomy.

Upon palpation there is no rigidity, and deeper palpation elicits no tenderness over any portion of the abdomen. More careful palpation fails to reveal the presence of any abnormal finding, as a tumor or a pulsation of any kind. The liver is not palpable, nor is the right kidney; neither is the left kidney nor the spleen. There is no tenderness and no tumor mass in the region of the gall-bladder and no tumor mass is palpable in the epigastrium.

As to the consideration of the question of plumbism, we have no lead-line; the blood examination is negative for degenerated reds; the patient has, it is true, abdominal pain with colic and also the presence of a constipation, but we cannot make a diagnosis of plumbism. Tabetic crises also must be excluded on the ground of the negative ocular findings; the pupils are equal and respond readily; the patellar reflexes are present, slightly exaggerated; the patient denies a venereal infection.

As to the existence here of some of the other possibilities: Aneurysm, for instance, must be excluded. The examination also fails to reveal a movable kidney, so a Deitl's crisis must be excluded. The absence of a history of pain, with negative physical findings in the region of the gall-bladder, likewise excludes a gall-stone and cholecystitis. The patient was admitted on the diagnosis of a kidney-stone, and as a result our attention was particularly directed to that organ.

The urine examined on four different occasions has remained constantly negative. A white count gave 8200. A cystoscopic examination was also made and revealed practically nothing. The ureters were catheterized and the urine from each kidney examined carefully, with negative results. An *x*-ray examination was also made on the 12th of the month for the existence of a kidney-stone, with a report that there is no evidence whatever of a renal calculus. Thinking that possibly we might have a specific infection, in spite of our negative history, and that possibly the pain could be accounted for on the basis of a luetic infection, a Wassermann was done on the 15th, but the report came back negative.

Searching still further for some explanation for these com-

plaints, the patient was given a test-breakfast on the 13th, but examination of the test-breakfast was normal—a normal acidity, no lactic, no blood, absolutely nothing but what might be found in a normal stomach. No blood in the stools. At a subsequent examination a second test-breakfast was also given, and the findings noted here are a slight increase in the mucus, a few red blood-cells, but the acid percentages are normal, and from this we are led to the conclusion that probably, so far as we can determine, at least from our laboratory reports, there is no gastric involvement, neither a peptic ulcer nor a carcinoma.

Inquiring still further into her history, and particularly in reference to the matter of the abdominal operation, we were interested to know for what reason this operation was performed and what was done. The operation was not performed in this hospital, so we haven't the advantage of a previously recorded history. We have only the statement of the patient. We have the evidence here, of course, of the laparotomy wound. A careful physical examination has been made on two different occasions, without detecting anything in the pelvis except possibly a slight increased resistance and tenderness on the left side. Evidently a supravaginal amputation was done with probably the removal of both tubes and ovaries. Perhaps the appendix was also removed. A rectal examination revealed nothing.

But the patient states that for some months after the operation she was comparatively well, and that several months later she began to have some abdominal distress and discomfort which has gradually increased. She has consulted various physicians, and she has received medicine from time to time to relieve her pain. On most occasions after the taking of medicine there was a decrease or temporary subsidence of the pain, and this was usually followed by sleep, so we naturally infer that she received some opiate. Is it possible that this patient is suffering from abdominal distress and discomfort for the lack of an opiate? She suffers from constipation, she has abdominal pain and distress, and it is possible that this is one of the factors in her present complaint.

Another possibility which we seriously considered and must take into account is the matter of a mucous colitis. This is a condition by no means infrequent. It occurs quite frequently in infants. In a second group of cases it is associated with pelvic diseases with pressure on the colon or rectum, with pain and the passing of a considerable amount of mucus. The third group includes the so-called "idiopathic" cases. The patient has attacks of constipation frequently alternating with diarrhea, and this condition is by no means uncommon after abdominal operations. She has at times a fair amount of mucus (and after the passage of the mucus in a typical case the patient is relieved), but this patient states that at the time of evacuation the pain is increased.

Up to this point, then, it was impossible to make any definite or absolute diagnosis, and, as a matter of simple routine and hoping to derive some further information, she was sent to the *x*-ray department, with the request that she be given a barium meal, particularly with the idea of determining, if possible, the presence or absence of abdominal adhesions following her pelvic operation; we thought that, as a result of the operation, adhesions might have occurred, and, with subsequent contraction, produced more and more distress, and that the condition today is due to adhesions in the lower portion of the abdomen. It is true that at no time have we been able to demonstrate peristaltic waves; there has been no abdominal distention, no fecal vomiting, and there is nothing in the symptoms strongly to suggest the presence of pelvic adhesions, but in the absence of any other evidence the request was made that a barium meal be given to ascertain what might be found.

The doctor has brought in the plates this morning with a report from the *x*-ray department. We will present these as they were taken. Patient received a barium meal, then seven hours later the first plate was taken, and you will observe that the barium is deposited in the lower portion of the stomach, with small masses located presumably in the upper portion of the stomach. The larger portion of the barium has passed on into the small intestine and the colon. The patient was immediately

given an additional barium meal, which brought out some interesting findings. The stomach was found extending well upward. The stomach shadow was definitely divided into an upper and lower portion with a marked constriction or narrowed band between. The interpretation by Dr. Blaine, our Röntgenologist, is that we have here an hour-glass constriction of the stomach. That little narrow, cord-like shadow connecting the two is the location of the stricture, with a pouch above and a pouch below. This third plate is the one taken a few minutes after the second one, and you can observe that we have practically the same condition. A fair amount of the barium has passed from the upper sac, through this constriction, and the lower portion has increased in size. A fair amount has already passed on through the pylorus into the small intestine. Dr. Blaine's interpretation of these shadows is that we have a positive hour-glass constriction, and it now becomes necessary to determine whether it is a temporary spastic constriction or an organic one, due either to an ulcer with a scar or a carcinoma. He proposes today or tomorrow to make another examination, and if no narrowing is present then we must conclude that it is simply a spastic constriction, but if it persists, and he is now quite definitely of the opinion that it will continue, then we have an organic or a permanent hour-glass constriction.

The presence of this condition brings up several rather interesting considerations. Is this stomach condition her primary trouble? Has she a stomach involvement and also a pelvic and peritoneal involvement? Or is the gastric disturbance only secondary, and the attacks of pain, colic, and vomiting due primarily to the peritoneal involvement? If the stomach is the primary involvement, then how are we to account for our gastric findings? We have had two test-meals, and both have revealed a normal finding. How are we to account for that?

In our history we mentioned, according to the statement of the patient, that two years ago she suffered from what was diagnosed as a gastritis. On close inquiry we are unable to elicit any symptoms that might lead to a tentative diagnosis of a gastric ulcer. Two years ago did this patient have a gastric

Fig. 27.—Hour-glass stomach. Showing stomach with upper and lower pouch, and narrow, constricted passage between. Seven hours after barium meal.

Fig. 28.—Hour-glass stomach. Taken soon after second barium meal. Upper pouch contains greater portion of barium meal.

Fig. 29.—Hour-glass stomach. Taken about twenty or thirty minutes after second barium meal. The greater portion has passed into lower pouch.

Fig. 30.—Hour-glass stomach. Taken two days later, immediately after giving barium meal. The upper and lower pouches persist and the connecting passage shows a definite sacculation.

ulcer and since that time cicatricial contractions producing an hour-glass constriction? If our further *x*-ray examination reveals a permanent constriction, then we must account for it on some basis, and we most naturally would conclude that the condition was secondary to a peptic ulcer and not to a carcinoma. The symptoms and findings are not indicative of a carcinoma. The further findings to be revealed by the secondary examination will be noted at a later date.

The prognosis depends, of course, upon the definite determination so far as possible of the existing condition; but at the present time we are strongly in favor of an exploratory laparotomy, even though our next *x*-ray report is negative; the hour-glass constriction having disappeared, then an exploration is indicated to determine the presence of peritoneal adhesions or whatever may be found. If the constriction persists after the next barium meal, then we have still more definite indications for surgical interference.

[NOTE.—April 5, 1915: A second *x*-ray examination was made two days later, and we have here again, as you can see, a very typical hour-glass constriction. The condition persisted with the second barium meal, the shadow being even more typical than in the first examination; here are the upper pouch, the lower pouch, and the constricted passage between. On the results of these repeated examinations the deduction was made that this was an organic constriction and not a spasmodic one.

She was operated last Thursday evening. As soon as the abdomen was opened it was found that the stomach presented a very definite hour-glass constriction, with a large upper and lower pouch and a well-marked constriction between. In searching for further findings it was discovered that the stomach was firmly attached to the lower surface of the left lobe of the liver. The adhesions between the stomach and liver were broken with some little difficulty, revealing a large opening into the stomach at the point of attachment. No evidence, at least macroscopic, could be obtained to justify the assumption that a neoplasm existed. The edge of the opening into the stomach

was firm, but there were no nodules or evidence of carcinoma. No attempt was made to do a resection, for the condition of the patient did not justify such a radical operation, but the upper and lower edges of the gastric opening were brought together by a double row of sutures crosswise to the long diameter of the stomach. No gastro-enterostomy was performed, for the reason that it would have been necessary to make the gastro-enterostomy so as to connect with the upper pouch, and that was not considered advisable. The patient since the operation has been on rectal feeding, and so far has had an uneventful career. What the subsequent course will be remains to be determined. No attempt was made to examine the abdomen or the condition in the pelvis; this was not thought advisable in view of the danger of infection from the gastric wound.]

CLINIC OF DR. WALTER HAMBURGER

Cook County Hospital

CONGENITAL PULMONARY STENOSIS. FREQUENCY OF ASSOCIATED TUBERCULOSIS

This patient (J. M.) is a Russian-Pole, twenty-five years of age, hotel porter.

He came to the hospital on the 23d of March with the following complaints: Sore throat, cough, difficulty in speaking, pain in chest, and weakness dating back three months.

For ten years he has had shortness of breath; he never had rheumatism or sore throat before the present illness. He has been at other hospitals before entrance here.

Family history: Father and mother both dead; one brother and one sister living and well. He is not married; he denies venereal disease. He drinks one to three beers a day and two to three whiskies.

The physical findings we will go over together. The most striking feature is his pallor; there is a slight flush in his cheeks; practically no cyanosis of ears or lips; he talks with a very marked, almost complete aphonia; he breathes a bit heavily. You can easily hear his difficulty in breathing.

On further inspection, particularly of his extremities, one notices the clubbing of his fingers; there is marked clubbing or widening and increased thickness of the ends of all the fingers. The same condition exists in the toes.

Looking a little closer, what do you see? He has an abdominal type of breathing with very little expansion of the chest. The supraclavicular spaces are markedly retracted, particularly on the right side. Otherwise his nutrition is fairly good; there is slight loss of weight; no jaundice; no marked cyanosis; good muscular development, and strikingly marked anemia.

On examining his eyes, his pupils are equal; they are small, symmetric; sluggish reaction to light and accommodation.

The teeth are in fairly good condition; slight pyorrhea present in both gums; tonsils are injected.

Neck: Sternomastoid muscles prominent; no palpable thyroid; no pulsating cervical vessels; no tracheal tug.

Chest: His chest is fixed to a certain extent, suggesting some emphysema; on percussion there is a decidedly diminished resonance in both apices, particularly the right apex.

Auscultation: Breath sounds practically normal throughout. He has some diminished expansion on the right side; posteriorly, hyperresonance occupying practically the entire lower lobes; he has fairly good excursion, particularly on the left. So much for the chest.

We forgot to speak about his pulse, which is normal, equal in both wrists, regular, good tension.

Heart: Cardiac dulness. We get the upper border of the liver dulness first; then, on outlining the heart, we find the upper border at the third rib; right border measures 4 cm. and the left border 11.5 cm. from the midsternal line. In other words, a practically normal right border, but a slight increase to the left.

His apex-beat is neither visible nor palpable; there is a palpable thrill at the base, especially in the pulmonic area, associated with a systolic murmur. On auscultation there is at the apex a rather loud systolic murmur transmitted slightly toward the axilla; it increases in intensity, particularly upward, progressively upward toward the right, reaches the point of maximum intensity in the second left interspace at the junction of the third left rib. In the pulmonary area there is a loud, rough, and irregular murmur (much louder and much rougher than usually occurs in a systolic mitral lesion). This murmur in the second left interspace is transmitted in all directions, but especially toward the left upper part of the chest. It is heard likewise in the aortic region, but not in the great vessels of the neck. It is heard all over the precordial area and transmitted over the entire region; it is heard only indistinctly in the right lower

Fig. 31.—Clubbed fingers. Pulmonary osteo-arthropathy.

Fig. 32.—Clubbed toes. Pulmonary osteo-arthropathy.

Fig. 33.—Skiagram of clubbed fingers. Pulmonary osteo-arthropathy. Note mushrooming of terminal phalanges and roughening of the sides of the shafts.

chest. The aortic area has the same systolic murmur; the second aortic can be heard with only slight accentuation. The second pulmonic sound is not audible. The systolic murmur can be heard in the back over the left scapular region, but with difficulty, and is not especially audible.

Examination of the abdomen: Neither liver nor spleen are palpable; there is nothing especially abnormal.

Extremities: With the exception of the clubbing of the toes, already mentioned, there is nothing especially abnormal; knee-jerks are present and active; slight emaciation.

Blood-pressure: Systolic, 120; diastolic 80; same, on both sides. Urine showed neither albumin nor sugar.

His throat has been looked at by Dr. Boot, with the following report—Larynx: Cords swollen; epiglottis swollen and reddened, infantile type; tuberculosis of the larynx. The sputum showed many tubercle bacilli.

His pulse has been regular, ranging from 70 to 86; respiration: 18, 20, 24; temperature: he has had slight afternoon rise.

Summary: The man has been sick for three months, showing, as far as the heart is concerned, a systolic murmur in the second left interspace, a palpable thrill, absent second pulmonic tone, left ventricle and probably right ventricle hypertrophied; clubbing of fingers and toes.

He had a fluoroscopic examination by Dr. Blaine on the 24th, which showed a dilated aorta.

DR. HAMBURGER: What is the matter with him?

VISITOR: He has tuberculosis of the right lung. He has a heart lesion; clubbing of the fingers and toes point to a heart lesion.

SECOND VISITOR: Pulmonary stenosis.

DR. HAMBURGER: If pulmonary stenosis, is it congenital or acquired?

VISITOR: From the history, it is acquired; ten years' duration.

DR. HAMBURGER (addressing Patient): How long have your fingers been clubbed?

PATIENT: About ten years.

DR. HAMBURGER: Pulmonary stenosis, if congenital, develops at birth, of course. It might show itself at puberty or on the development of the individual. However, pulmonary stenosis is practically always congenital. Acquired is so rare that it does not come in for consideration. What else may it be besides pulmonary stenosis?

VISITOR: Persistent ductus arteriosus.

DR. HAMBURGER: The strongest point of differentiation in this case between persistent ductus arteriosus and pulmonary stenosis is the absence here of the second pulmonic sound. It speaks for a lesion of the pulmonary valve. But, on the other hand, most of the cases of pulmonary stenosis are cyanotic; at birth the baby is often blue; the patients are deeply cyanotic even up to adult life; the eyes are watery; the whole picture is marked by an intensely blue color of the patient, "morbus cæruleus" (blue sickness). You have seen them on the street. These persons are the typical individuals suffering from congenital pulmonary stenosis. This man has no cyanosis. He has most of the symptoms of congenital pulmonary stenosis, but he is not cyanotic.

It is pretty difficult to differentiate closely between congenital heart lesions; it is an unprofitable thing to do. The evidence in this case is that it is a congenital pulmonary stenosis, with the exception of cyanosis. It is an unusual case in this regard. Stenosis of the pulmonary orifice is the most frequent congenital lesion, but it is nevertheless extremely rare.

Now, there is one other point: Pulmonary tuberculosis is common with pulmonary stenosis, either congenital or acquired. On account of the poor circulation in the lung the tubercle bacillus finds a favorable place to propagate. This is particularly interesting in contradistinction to the absence of pulmonary tuberculosis in cases of mitral stenosis, where, owing to the passive congestion of the lung with a constant hyperemia, tuberculosis is said to be very infrequent and is seldom found.

Likewise, the clubbed fingers; while they are present in various chest troubles, they are classically present in tuberculosis. This condition of clubbed fingers is spoken of technically

Fig. 34.—Skiagram of clubbed toes. Pulmonary osteo-arthropathy. Note mushrooming of terminal phalanges and roughening of the sides of the shafts.

Fig. 35.—Skiagram showing fibrosis of both right and left upper lobes (fibroids, pulmonary tuberculosis). Heart shadow increased to right and left, mediastinal shadow increased in width.

as secondary hypertrophic osteo-arthropathy. Locke, of Boston, has recently studied 5 typical cases of hypertrophic osteo-arthropathy and 39 of simple clubbing of fingers and toes occurring in a miscellaneous group of diseases, arriving at some rather interesting conclusions. In particular, he points out that osteo-arthropathy is always a secondary disease, and that pulmonary tuberculosis is probably the most important primary disease, though rarely inducing the extreme degree of bone and joint changes seen with bronchiectasis. He points out further that simple clubbing of fingers and osteo-arthropathy should be considered as identical, the former representing the early stage of the latter.

We have had skiagrams made of the hands and feet of this patient. Dr. Blaine's report is as follows:

"*X*-ray examination of hands and feet show marked clubbing of the ends of fingers and toes, with mushrooming of the terminal phalanges and a roughening of the sides of the shafts of the phalanges. Typical pulmonary osteo-arthropathy."

Another finding which may be present is polydactylism, or any other evidence of congenital maldevelopment. The only point in this case which is suggestive at all is the note by Dr. Boot, "epiglottis, infantile type"; this may be a slight indication of a congenital anatomic deformity, the same as six toes, etc.

The *x*-ray pictures: Fluoroscopically, as has been said, there was a diffuse widening or dilatation of the aorta. The skiagram showed a marked fibrosis of both upper lobes, particularly on the right side; there is an old fibrous tuberculosis. The heart shadow may be seen increased to the right and to the left, and the mediastinal shadow is wider than normally.

In a typical pulmonary stenosis there are few, if any, changes in the heart outline. In persistent ductus arteriosus there is a marked change in the shadow of the pulmonary artery. You will see it here (referring to drawing in student's text-book); an increase in size and pulsation of the pulmonary artery is due to the following: In a persistent ductus arteriosus with the increased pressure in the aorta you get an increased intrapul-

monary blood-pressure, and as a result dilatation and hypertrophy of the pulmonary artery. Now, that dilatation and enlargement of the pulmonary artery may be mistaken for aneurysm. In a case where it is difficult to interpret the presence of a dulness in the left second and an enlarged pulsating pulmonary artery, it speaks strongly for a persistent ductus arteriosus. If he had dulness there and the aneurysm you would be inclined to think he had a persistent ductus arteriosus.

This case is interesting from the standpoint of differential diagnosis. When he came to the hospital he was admitted as a suspected aneurysm, largely on account of aphonia, brassy cough, and substernal dulness. The *x*-ray findings apparently confirm this, at least that he had a dilatation of the aorta. However, more careful examination, particularly with reference to the systolic murmur in the second left interspace, palpable systolic thrill over the same area, as well as the finding of pulmonary tuberculosis and clubbed fingers and toes, makes one fairly certain that we are dealing here with a congenital pulmonary stenosis. As we have said, the only point against this diagnosis is the absence of cyanosis, but, in spite of the statement found in most text-books, it is probably true that a large majority of these congenital heart lesions may not have a high-grade cyanosis, if any.

ANEURYSM OF THE ARCH OF THE AORTA AND OF THE ABDOMINAL AORTA. THE RÔLE OF SYPHILIS IN THE ETIOLOGY

In going over this man's history very briefly we find he is a gas-fitter by occupation, fifty-one years of age, and states that he has had heart trouble for two or three years; was in Cook County Hospital one year ago complaining of symptoms similar to those of the present time. Shortness of breath for last two years or more; can hardly walk up stairs on account of it. Cough

for many years, excruciating at times, severe for about six months; never coughs or spits up blood. Palpitation of heart noticed about five years ago; asthmatic attacks for last six months, present most of the time; feels as if he were smothering and needs more air. Headache, frontal type. Spots before eyes; he has noticed this for about ten years; spots look like a screen going by. Pain in chest has been noticed off and on for four years. Pain in the epigastrium during last five or six months; this is worse when he coughs a great deal. No urinary disturbance.

Previous illness: Was in Cook County Hospital with similar trouble four years and one year ago respectively.

Habits: Drinks ten or more beers daily and five or six whiskies a day. Venereal: Gonorrhea in 1892; chancre in 1892. Treated six months.

Family: Father dead, heart trouble. Mother dead, heart trouble. Two sisters and two brothers living and well.

Physical examination: Well nourished, wheezing, considerable dyspnea; does not appear very ill. Eyes: Pupils rather small. Mouth: Several decayed teeth; pyorrhea marked. No palpable glands in neck.

Chest is barrel shaped; expansion slight. Lungs: Impaired resonance over right apex anteriorly and over both apices posteriorly, and especially near the base throughout both lungs. Heart: Apex 1 inch outside nipple line; right border $2\frac{1}{2}$ inches outside midsternal line. Soft systolic murmur over precordial area; greatest intensity at apex, transmitted to axilla; arhythmia rather marked; pulse tension increased; moderate arteriosclerosis.

Abdomen: Liver four fingers below costal arch, rather tender, smooth; no evidence of fluid; spleen not palpable. Genitals normal. Extremities normal. Reflexes: Knee-jerks exaggerated; no Babinski. Blood-pressure, systolic 170, diastolic 85.

As he sits there he feels perfectly comfortable as far as pain or dyspnea are concerned. His color is fairly good, his face is a little flushed, his lips and ears are not cyanotic, his skin is moist and warm; his hands are perfectly negative, are not cyanotic,

and his fingers are not clubbed as in some of the cases of congenital heart disease we have seen; no clubbing of toes.

On inspection there is remarkable pulsation seen in the suprasternal notch. You see the pulsation is irregular.

DR. HAMBURGER: Do you see anything else?

VISITOR: Wide epigastric angle.

DR. HAMBURGER: Do you see anything else?

VISITOR: Pulsation in the vessels of the arm at the elbow.

DR. HAMBURGER: Yes, rather marked pulsation. He has a slight flaring out of the lower intercostal margin.

His chest shows little expansion. (Take a deep breath.) You can palpate this mass in the suprasternal notch, and on palpation you will find marked irregularity. There is some slight pulsation on the left; there is no palpable thrill; there is no definite heaving impulse.

On percussion: His upper right chest is dull; likewise his right apex. There is a slight substernal dulness, which extends a little bit to the left. His left heart extends to the left of the nipple line.

On auscultation what do you hear? A systolic murmur at the apex and over the base—both extremities of the sternum. *The apex-beat is at the fifth interspace, just outside of the nipple line.* Soft systolic blow at the apex, transmitted to the left; likewise a blow at the base of the heart, soft, systolic in time. A louder, systolic bruit in the suprasternal region; there is a difference in the quality, especially in the loudness and roughness of this murmur. He has some tortuosity of the blood-vessels, or hardening. What do you think of him?

VISITOR: Mitral stenosis.

DR. HAMBURGER: Why?

VISITOR: Systolic murmur at the apex.

DR. HAMBURGER: Are mitral stenosis murmurs systolic in time?

VISITOR: No, I don't mean that; mitral insufficiency.

DR. HAMBURGER: Has he anything else?

VISITOR: I don't know whether it is an aneurysm or not.

DR. HAMBURGER: What do you think?

ANOTHER VISITOR: I think it is an aneurysm of the transverse portion of the aorta.

DR. HAMBURGER: Why do you think so?

VISITOR: Because it comes so high up in the neck, although it is not so large as some I have seen.

DR. HAMBURGER: What do you think of the murmur at the apex?

VISITOR: I think that is a mitral insufficiency.

DR. HAMBURGER: Then he has two things?

VISITOR: Yes.

ANOTHER VISITOR: Has he had syphilis?

VISITOR: Yes.

DR. HAMBURGER: What about the pupils in aneurysm?

VISITOR: They may be dilated or unequal and irregular.

DR. HAMBURGER: On continuing the examination a little bit further, he has a distinct systolic tracheal tug which, however, is not present with every pulse-beat. His pupils are unequal; they react to light and accommodation. I think you will see the left pupil is just a bit larger than the right; at different times it is more pronounced; they are symmetric. His pulse is extremely irregular. He has not much cough. There is nothing of special moment in his lungs or abdomen. He has no ataxia.

The *x*-ray examination is as follows: The skiagram shows a wide aortic shadow suggesting a large aneurysm of the arch. This merges with a marked widening of the mediastinal shadow which displaces the trachea to the right. The left ventricle is increased in size. There is diffuse dilatation of the aorta extending downward to the diaphragm. There is considerable increase of tissue in the middle and upper right lobes of the lung.

The question arises, Is this a mediastinal tumor or an aneurysm?

Blood examination showed nothing abnormal. Urine examination showed trace of albumin and granular casts.

Then to summarize in this case: First, he has had symptoms dating back three or four years; smothering spells which he called asthma; some pain in the chest, but not striking. From

the findings on physical and *x*-ray examinations the conclusion is practically clear that he has aneurysm.

The points speaking for aneurysm definitely in this case are: first, dulness; second, the pulsation at the suprasternal notch; third, tracheal tug. You can add to that the systolic murmur.

In analyzing the other findings, his symptoms resemble much more the symptoms of a myocardial breakdown than of aneurysm. Everything that he complains of practically can be ascribed to his myocardium. His shortness of breath, palpitation of the heart, asthmatic attacks, are all evidences of myocardial disease or cardiac decompensation. So that, when examining a heart, unless you bear in mind the possibility of aneurysm, it may be overlooked.

DR. HAMBURGER: How about the high blood-pressure?

VISITOR: It may be due to cardiac insufficiency, but it may likewise be due to disease of the kidney. With a man of his history—syphilis, alcoholism, high blood-pressure, and the heart out to the left—you are entitled to a diagnosis of chronic interstitial nephritis.

DR. HAMBURGER: How about the other symptoms?

VISITOR: The soft systolic blow at the apex may be due to mitral insufficiency with slight dilatation of the heart, and not to myocarditis. As far as aneurysm is concerned, the history of syphilis is of importance because of its association with aneurysm.

DR. HAMBURGER: His larynx was examined, and it was found that his left vocal cord does not move; that it is paralyzed.

The aneurysm is located on the left. The difference in the pupils, slight increase on the left side, which is a frequent finding in aneurysm, may be due to pressure on the left sympathetic, but we have no clear evidence of that.

Why are we interested in his knee-jerks?

VISITOR: Tabes.

DR. HAMBURGER: The association of tabes and aneurysm is not so very infrequent. It is not uncommon to find a tabes associated with aneurysm, and often the eye-signs of aneurysm are the eye-signs of tabes. In this man they are the signs of aneurysm and not of tabes.

Fig. 36.—Skiagram of aneurysm of the arch of the aorta, showing a wide aortic shadow, with displacement of the trachea to the right.

Fig. 37.—Skiagram showing displacement of stomach by large abdominal aneurysm.

Treatment.—Several things, of course, are to be taken into consideration. First of all, myocarditis with heart muscle insufficiency; that is, early heart failure; in myocarditis with early heart failure give the patient rest in bed with restriction of fluids, digitalis, and, if necessary, an ice-bag to the precordium; fluid limited to about 3 pints. This case should be treated as any case of heart failure irrespective of the presence of aneurysm. Then, later on, as the heart muscle is improved and the rapid pulse slowed under digitalis and rest, special treatment may be directed toward the aneurysm. Medical treatment is unsatisfactory; you can't do a great deal; the classical treatment is rest and restriction of fluid and food.

Tufnell's treatment of aneurysm consists in restriction of fluids and food, a relative starvation or fasting of the patient, so that the blood-pressure is reduced to a minimum. Then the ice-bag to the precordium, supposedly to act as a constricting method; potassium iodid, particularly in syphilitic aortitis, and for its specific action on the blood-vessels.

Then, of course, the question of surgery is often discussed, and sometimes surgery is undertaken in aneurysm, but so far has been most unsatisfactory as a whole, and nothing can be expected with the present technic—the insertion of gold wire or platinum wire into the interior of the anuerysm, coiling up the wire to produce shrinkage of the sac by the deposit of fibrin.

Another point with this man is that, in addition to aneurysm, he has kidney involvement with high blood-pressure. We shall attempt to lower the blood-pressure as far as possible by prohibiting anything which will continue the high pressure—restricting protein somewhat, restricting meat extracts and meat soups entirely, cutting down the total quantity of food, giving him only food in small amounts at a time, with elimination of salt from his food throughout. But, in spite of all these things, aneurysm is a progressive disease. Complete rest in bed and restriction of food is the best treatment for these patients.

This is rather an easily diagnosed case. There is just one point which I do want to speak of; that is, the question of pain in aneurysm. All of his symptoms are symptoms of broken

compensation. He makes no complaint of pain except of a slight one in the chest. Now pain is usually one of the most frequent and constant complaints. Pain of various kinds, and particularly pain that is constant, deep-seated in character, often worse at night. Pain, as distinct from other symptoms, is characteristic of aneurysm.

DR. HAMBURGER: How much pain did you have?

PATIENT: Well, quite a bit of pain in the chest; more shortness of breath and more of the asthma.

This case is exceptional in that respect—the relatively slight pain. In any case where you suspect aneurysm it is well to inquire about the pain. In any individual who comes to you complaining of pain the question of aneurysm should be in your mind.

DR. HAMBURGER: Where is your pain?

PATIENT: In the chest.

DR. HAMBURGER: All the time?

PATIENT: Down my left arm; it has been there now three or four months.

DR. HAMBURGER: There all the time?

PATIENT: Yes.

DR. HAMBURGER: What kind of pain have you in your heart?

PATIENT: Dull pain; not stinging pain.

DR. HAMBURGER: Is it worse at night or day?

PATIENT: Worse at night.

DR. HAMBURGER: Is it getting worse right along?

PATIENT: It was worse when I was home.

DR. HAMBURGER: That pain he speaks of is characteristic of angina pectoris. This man has pain starting in the chest and radiating down the arm.

VISITOR: Has a Wassermann test been made?

DR. HAMBURGER: A Wassermann was made which was four plus. Since he has been in the hospital he has had repeated mercury rubbings and potassium iodid.

The next patient is a colored woman, forty-three years of age, American. Came into the hospital complaining of dyspnea, inability to lie down, swelling of ankles and feet. Rheumatism

four years ago, which cleared up and has not bothered her since. About one month ago she had choking or smothering spells at night located around the heart; spell would last a few minutes and then be followed by profuse sweating. Menopause one and one-half years ago; no spells at that time.

Dyspnea on exertion, becoming worse each day; at present unable to lie flat; patient is propped up on pillows. Edema of ankles since last week, more marked at night. Bowels and urination normal, appetite normal, weight normal. Patient had rheumatism four years ago; also the diseases of childhood.

Parents are dead. Mother died of grip; does not know what father died of. She has no brothers or sisters.

Menstruation began at the age of thirteen, has always been regular, and stopped at the age of forty-one. Had one child, which died at the age of five years of spinal meningitis. Had no miscarriages. Denies venereal disease. Uses no alcohol.

Examination.—Pupils equal and regular. Tonsils not enlarged. Tongue heavily coated. Teeth: Marked pyorrhea and many teeth decayed.

Chest: Nutrition poor. Visible pulsation of left chest, especially lower part. Systolic retraction of the third and fourth interspaces, left side, following a preliminary bulging. Resonance normal over lungs. Inspiratory crackling râles over lower half of left side posteriorly. Lung borders, posteriorly, normal; excursion poor. Dyspnea.

DR. HAMBURGER: Is your husband living?

PATIENT: Living and well; strong, robust man.

DR. HAMBURGER: Now the most striking thing in this patient is this mass in the abdomen, a localized mass about the size of an apple; shows strong, regular pulsation, which on palpation seems to be systolic in time. This tumor-mass moves somewhat on respiration, has a very marked expansile pulsation, and is extremely tender.

On auscultation over this mass you can hear a distinct systolic bruit. It is perfectly clear that this is an aneurysm of the upper portion of the abdominal aorta. This is a rare case; the rarity is increased by the fact that it is in a woman.

Aneurysm is particularly a disease of the colored race. In going through this hospital, for example, you will find aneurysm tremendously higher in the colored race than in the white. The frequency of lues in the colored race is also very high.

Aneurysm of the abdominal aorta is simply interesting from the standpoint of rarity.

It is, however, likewise interesting from the standpoint of differential diagnosis; epigastric pulsation is a frequent finding in many normal individuals, without, however, a demonstrable tumor pulsation. This marked epigastric pulsation is seen particularly in women, frail, thin women, in neurasthenic men and women, in anemic patients; and in those patients abdominal aneurysm is frequently suspected and even frequently diagnosed. The vast majority of these cases, of course, are simply a normal pulsating abdominal aorta and not aneurysm.

I have tracings here, but they do not show anything special.

She has no history of specific disease. She has had no miscarriage. Wassermann test was made, which was strongly positive.

DR. HAMBURGER (to Patient): How long have you noticed that mass there?

PATIENT: Ten or twelve years.

DR. HAMBURGER: Has it been getting larger?

PATIENT: Yes, all the time

DR. HAMBURGER: Is there any pain connected with it?

PATIENT: No, only when you press on it.

DR. HAMBURGER: Getting worse all the time?

PATIENT: Yes, my breath is short.

DR. HAMBURGER: You can see that it is getting more prominent.

PATIENT: Yes.

DR. HAMBURGER: How did you notice it first?

PATIENT: I went to a doctor; I had a pain. On physical exertion I had pain; the doctor said I strained myself.

DR. HAMBURGER: *X*-ray shows a displacement of the bismuth shadow of the stomach so that it appears as a circular mass.

Let us now recapitulate the principal phenomena of aneurysm.

The symptoms can be divided into:

(1) Functional. Palpitation is a finding in aneurysm, likewise fainting (syncope), and, of course, pain, particularly the anginal type of pain.

(2) Symptoms due to pressure: Pressure on the phrenic nerve, causing hiccup; pressure on the sympathetic, either right or left, causing anisocoria (difference in the size of the pupils), flushing and sweating; pressure on the laryngeal nerve, causing aphonia, paralysis of the vocal cord, cracked voice, brassy cough. Pressure on the esophagus, causing dysphagia. Pressure on the trachea or bronchi, causing cough, dyspnea, orthopnea. Pressure on the lung, giving symptoms of atelectasis. Pressure on bronchi, giving symptoms of putrid bronchitis, pulmonary tuberculosis. Pressure on blood-vessels, causing hemoptysis.

Outline of the physical signs: The physical signs are more important than the symptoms.

On inspection you may get pulsation of the chest rather frequently which is not caused by aneurysm. Now, it is important to distinguish the place where you get the pulsation and to differentiate between a tumor and an aneurysm.

First, you may get a diffuse pulsation of the chest in aneurysm. You also may get diffuse pulsation in various blood diseases, goiter, etc. Second, a heaving impulse over the entire chest. Third, aneurysmal tumor. With diffuse pulsation you may also get an expansile pulsation.

Let us speak a minute of the points of pulsation in a normal chest: At the suprasternal notch you see it very frequently in the normal individual. You normally get pulsation at the apex; you get epigastric pulsation; you often get pulsations due to a little dilatation. Then there is the tracheal tug, difference in pupils, etc.

Then on inspection of the face: There are no special signs except flushing and sweating. You may get cyanosis; not the cyanosis due to broken compensation or congenital lesions, but cyanosis due to pressure, which prevents venous return, a mechanical process.

Treatment.—Some get well. The accidents in aneurysm are those of rupture or perforation of the neighboring structures, causing hemoptysis. Some patients live for a good many years in spite of a threatening or formidable aneurysm. The prognosis is extremely hard to give. In certain cases of aneurysm, in which there is urgent distress and the possibility of rupture, blood-letting is an absolute necessity and gives good results. The mechanical withdrawing of blood is followed by relief.

Aneurysm is a fairly common disease, and you will meet it more frequently than you believe. Contrary to the statements in some of the text-books, aneurysm may occur in any sort of an individual.

CLINIC OF DR. RALPH C. HAMILL

COOK COUNTY HOSPITAL

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

Varieties:

- Vascular and perivascular.
- Early inflammatory:
  - Meningitis.
  - Encephalitis.
  - Myelitis.
- Late:
  - Gumma.
  - Sclerosis.
  - Thrombosis.
  - Softenings.
- Parenchymatous:
  - Paretic dementia.
  - Tabes dorsalis.
  - Pyramidal tract degeneration.
  - Anterior horn cell disease.
  - Optic atrophy.

Inasmuch as syphilis presents so many varieties and is the cause of so many diseases of the central nervous system, I thought it would be well to go over the subject and show a case that presents both the early and late symptoms of syphilis of the central nervous system. The varieties of clinical expression of syphilis of the nervous system I have put in this scheme, which is a combination of clinical and pathologic entities and syndromes, and I have divided the cases primarily into parenchymatous and vascular, including perivascular. At one time it was denied that there was such a thing as parenchymatous syphilis, but the finding of the spirochete in the nerve-cells, in the posterior columns, and in the anterior horn cells, with the destruction of the structures in which the spirochetes

are found, does away with the idea that syphilis does not attack the parenchyma of the nervous system.

It was formerly thought that syphilis acted entirely upon the blood-vessels and meninges, but now I believe we are justified in including in the forms of syphilis of the central nervous system paresis with involvement of the cellular elements and association fibers of the cortex, with some predilection for the frontal regions of the brain, tabes dorsalis with the involvement of the posterior roots and posterior tracts of the spinal cord, degeneration of the pyramidal tract as evidence of direct action by the spirochete, anterior horn cell disease with typical progressive muscular atrophy findings, and optic atrophy as the expression of the invasion of the optic nerve by the spirochete.

Of the vascular and perivascular types there are the early and inflammatory forms, of which there are two varieties, namely, those due to involvement of the blood-vessels within the substance of the brain and spinal cord and those due to involvement of the blood-vessels of the meninges.[1] Then there are the late forms, softenings, that occur as the result of the closure of blood-vessels in the brain and spinal cord, and the meningomyelitic forms, due in all probability to the spreading of the toxins combined with an anemia of the peripheral zone of the spinal cord.

Case I.—This patient, as the history will show, suffered from the early changes of syphilis many years ago, and because of improper care or because of the incurability of the disease now suffers from the late manifestations. The history is somewhat faulty because he is not perfectly sure of all his data—says his memory is not as good as it used to be—but he says that he had double vision thirty years ago, which came on a month or so after he had some "ham-colored spots" in the palms of his hands, and he took some mercury for about three weeks at that time.

He is sixty-four years of age, married, a newspaper writer, and entered the hospital on the 2d day of March, 1915. There were two children born, both within the first three years after

[1] The changes may be widespread and diffuse or, after the very early stages, localized and more or less discrete—gumma.

marriage, in all probability before he got this infection. Those two children are now living and well. He had gonorrhea several times before marriage. The exposure which he thinks is responsible for this present condition took place in 1884—thirty-one years ago. The attack of double vision was the first evidence of involvement of the nervous system. According to the history this symptom lasted only a short time.

Then seven or eight years ago his eyes became crossed again. So far as he is able to tell us, both these attacks have been without a great amount of headache. He worked up to two years ago and then began to feel weak in the legs. This weakness was without any great amount of stiffness or spasticity. He felt as though he would have to carry a cane; was uncertain on his legs, says he was not sure of their support. Then ten months ago, after sitting for a while he got up and started to walk; his legs suddenly gave way under him and he has not been able to walk since.

About two years ago he began to have difficulty in urinating. This has gradually increased, so that now he is entirely unable to control his water. It runs from him practically all the time, and he has now developed a cystitis.

To analyze this history: The early attack thirty years ago; then a year or so after the infection appeared the early expression of syphilis of the central nervous system, coming under the head of the early inflammatory vascular and perivascular manifestations. In all probability it was an affair of the meninges.

This conclusion is arrived at because of the following considerations: Vascular and perivascular involvement of the meninges at the base of the brain is an exceedingly common early manifestation of syphilis of the nervous system. The oculomotor nerves—the third, fourth, and sixth—run forward along the base of the brain. The fourth and sixth run a long course through the basilar meninges, less true of the third; at any point of their course they are exposed to the disease processes spreading from the meninges. There are other factors to be considered in explaining the frequency with which diplopia

is met with in the early history of nervous syphilis. Three of the total of twelve cranial nerves have to do with the extrinsic eye muscles. Disturbance of their function (confusing double vision) is much more readily appreciated by the patient and remembered much more easily than disturbances of smell, hearing, paresis of the tongue, etc. In getting a history of an early syphilis the diplopia is the thing to get information on pre-eminently.

Then this man went along for twenty-two years without any difficulty, without knowing that he was harboring an infective agent, a disease-producing microbe, and then—seven or eight years ago—he again has diplopia, which, in all probability, was the expression of the late involvement of the vascular system, occurring as a more or less sclerotic process, and a general involvement, at the base of the brain, of this nerve a second time. We don't know which nerve was caught the first time, but the probability is that it was the sixth nerve because of its long course at the base of the brain; and, second, because syphilis often attacks the same place in the later that it did in the earlier process.

So eight years ago he again got an involvement which produced diplopia, and then for six years that was the only symptom until he commenced to have this difficulty with his gait. Now this disturbance of his gait is difficult to analyze because it is hard to get an exact description of it, but late syphilis produces two types of disturbance in locomotion: The tabetic type, the ataxic, the disturbance of gait due to the fact that the central nervous system is no longer informed of the exact state of the muscles of locomotion, and, consequently, doesn't know just how to direct their use; that's perhaps the commonest form. The less common form is due to the involvement of the pyramidal tract. The two types of gait are distinctly different, and usually we can determine by the history which type we have to deal with. That is, the tabetic or ataxic type is characterized by the fact that it is more difficult for the patient to walk in the dark because he can't use his eyes to control the movements of his legs. Second, he complains of the fact that it

is more difficult for him to go downstairs than to go up; the reason is that in going down stairs the step below is so much further from the point of vision than the one that he is leaving. Also when one steps forward one loses sight of the foot which is left behind. It is the one which is doing the work that is lost sight of, so the tabetic does not know just what the state of the muscles in that foot and leg is; consequently, in going down stairs he has the feeling that he is falling down stairs all the time, and he does fall from one step to another; whereas, in going upstairs the leg which is doing the work is in the front and is kept in front all the time. These two factors are the most common given in the history of a tabetic gait.

Of the spastic gait the common account given by the patient is that he commences gradually to notice a stiffness in his legs which comes on after he has been exercising for a while. The history usually reads somewhat in this way: When I start out to walk I am all right, but after walking two or three blocks or a half mile my legs commence to get stiff and they feel as if I cannot move; they commence to stick to the ground and I have to rest for a little, and after resting I can go on as well as ever, but soon the stiffness comes back again. That is what is commonly referred to as *intermittent claudication*, and is due to an interference with the supply of nutrition; to a thickening of the blood-vessel walls along the course of the pyramidal tract. These blood-vessels are able to supply sufficient nutrition for a short time, but they are unable to keep up a continuous effort and continuous supply. This intermittent claudication gradually becomes a permanent symptom owing to degenerative processes going on in the pyramidal tract because of insufficient blood-supply, or any of the sclerotic vessels may become thrombosed and so cut off the motor impulses passing to the body below the point of lesion.

Now this man says that his legs started to become weak about two years ago. They showed about the same degree of weakness for a year and then gave way and let him fall, and since that time he has been unable to walk. So we have to assume syphilitic thickening of the arterioles of the cord with eventual

thrombosis and cutting off of the downward flow of motor impulses.

In the examination of this man you see that the great toe is held up all the time; the tendon of the extensor is very prominent; it stands up, has a marked rigidity. That in itself is the equivalent of the Babinski sign. It often happens in these spasmodic conditions that any movement of the great toe is prevented. It is held rigidly in its posture or there is a slight increase of extension. You see there is some slight increase. The other foot is the same way, with a slight increase in the extension. There are contractions of both legs holding them in this position, flexed at the hips and knees to right angles. The hamstrings, as you see, are quite rigid and the deep reflexes are all very much increased. It is quite difficult, however, to demonstrate the fact that they are increased, or even that they are present, because at the same time that the tendon is struck to demonstrate the reflex you also get a contraction of the antagonist, with the consequence that the joint is fixed and does not allow the characteristic movement that we get in the reflex. There is a reflex there, and if we could get a little relaxation, as sometimes we do, we could show that there is a clonus, but it is impossible to do this now. You see quite a sharp reflex here of the quadriceps tendon, but this is also associated with a jerk of the hamstrings. Consequently, there can be no movement of the lower leg as is ordinarily seen in the patellar reflex.

Of striking importance in this man is the fact that any stimulus applied to his legs produces movement which is reflex. That is, you see that any touching of that leg, any pressure applied to any group of muscles or any portion of the foot or leg, will produce a reflex movement. Where the stimulus is strong, you get a strong movement; where it is weak, you get a weak movement. There is, consequently, a condition that might be spoken of as hyperesthesia. That is, you get this sharp withdrawal, this violent reaction, of the legs to any stimulus, but there is no *true* hyperesthesia. Any stimulus applied to the lower extremities produces a reflex in the spinal cord because of the

cutting off of the upper neuron, of the balancing and restraining force of the upper neuron system.

Sensation in the legs seems to be fairly normal. The arms are in fairly serviceable shape. The finger-to-nose test is fairly well performed. You see there is a slight swaying, but otherwise it is done all right; there is no intention tremor, such as we see in multiple sclerosis, and no tendency to hit any other part of the face, but you see there is a little bit of unsteadiness. It is not a perfectly smooth movement such as a normal person can readily execute. There is a little jerking, as if the mechanism were unbalanced—as if the mechanism were disturbed. The deep reflexes in the arms are very brisk; in fact, there is almost a clonus in the forearm on the right side following the percussion of the radius near the insertion of the supinator longus.

There is marked diplopia with strabismus due to paralysis of the sixth nerve on the right side. There is no involvement of the jaw reflex. There is no difficulty in swallowing. The abdominal reflexes are diminished, especially on the right, where they are practically absent. On the right side, where there is the marked wrist-jerk, we find a loss of the abdominal and cremasteric reflexes. The pupils react perfectly to light and accommodation. The bladder function, as I have said, is very markedly interfered with; the sphincter reflex is gone. Although he suffers from constipation, there is sometimes an involuntary evacuation of the bowels. Associated with these symptoms there is a decided loss of sensation over the area of skin supplied by the third and fourth sacral segments; a small area on either side of the buttocks and running down a short distance toward the dorsum of the thighs. In this area, pain, touch, and temperature sense are all diminished; there is a distinct difference between sensation there and on other parts of the body. Sensation in the legs is fairly well preserved.

I think I have gone into the early symptomatology sufficiently; the early diplopia being due to the catching of the sixth nerve on the right side in some inflammatory zone of the basal meninges. The first one of his late symptoms is of the same nature, and, in all probability, located in the same place attacked

by the earlier lesion. The next symptoms are those of involvement of the pyramidal tract, in all probability due to a diffuse thickening of the small arterioles supplying the spinal cord. The pyramidal tract extends from the Rolandic cortex of the brain in an unbroken column down through the internal capsule, the pons, the medulla, and down through the lateral column of the spinal cord to its lowest segments. It is vulnerable at any level of its long course, and the shutting off of the blood-supply at any level entails a failure of passage of motor impulses beyond this level. Hence, in vascular disease of the spinal cord symptoms of involvement of the pyramidal tract are very common.

The involvement in this man shows itself in marked spasticity, not paralysis, because, as you see, he does move his legs, voluntary movement is there; he can move his toes and ankles, and has a very slight movement in his knee- and hip-joints, but the marked spasticity prevents any degree of freedom, and that spasticity is due to the involvement of the pyramidal tract. The bladder symptoms, which are very distressing, are, in all probability, due, in part at least, to this involvement of the pyramidal tract. Secondarily, however, they are due to a local involvement of the sacral cord. That is, there is loss of the anal sphincter reflex, there is a loss of sensation in the distribution of the third and fourth sacral segment, and there is the interference with the bladder reflex. These symptoms refer to the local involvement of the second, third, and fourth sacral segments; probably in the nature of a meningomyelitis.

Such a clinical picture as this man presents must be diagnosed from that presented by amyotrophic lateral sclerosis, from that presented by multiple sclerosis, and from that presented by arteriosclerosis. Amyotrophic lateral sclerosis running a course beyond two years practically always produces more atrophy than we see in this man. It is associated with fibrillary twitching—something that we have not seen in this case. In amyotrophic lateral sclerosis there is no involvement of the sphincters and no sensory disturbance. On the other hand, sphincter and sensory disturbances and diplopia, all common in syphilis, are striking symptoms in this patient.

Multiple sclerosis is a disease of early adult life, coming on in the third or fourth decades. This man's disease comes on in the sixth decade. Multiple sclerosis has, in those cases which are at all advanced, marked nystagmus and intention tremor. These symptoms are not prominent in this patient. In multiple sclerosis the history is one of more or less constant involvement, with periods of temporary improvement and a disability coming on in the course of four or five years at the outside, whereas this man's first symptoms are separated by twenty-five years at least from his latest symptoms, and in the interval he is, to all intents and purposes, well.

Arteriosclerosis, such as occurs in Bright's disease and in rheumatoid conditions not on a syphilitic basis, would show more gross changes. I mean by that, focal changes of a grosser nature. There are focal changes in this man of the sixth nerve, and of the bladder and anal sphincters, but there is nothing in the history that we can see that would at all point to a sudden giving way of a large blood-vessel. On the other hand, there is undoubtedly in this man an arteriosclerosis, but on a syphilitic basis, and a sclerosis which so far as the present symptoms are concerned seems to involve the arterioles, the smaller rather than the larger vessels, so we cannot really say that this is not an arteriosclerosis. It is an arteriosclerosis, but of a syphilitic type.

Now, besides the clinical manifestations that we can see in this man, and the symptoms that lead us to diagnose it as syphilis, the spinal fluid shows 32 cells to the cubic millimeter and an increase in the globulin. A Wassermann has not yet been made, but we can take the findings that we already have as very strong evidence supporting our diagnosis of syphilis.

The prognosis in this man is bad, of course. He has changes in the small blood-vessels, which are practically obliterated, and these small blood-vessels are end-arteries supplying the spinal cord along the course of the pyramidal tract. The early symptom of diplopia that came on a year or so after his infection was at that time a curable symptom because it was due to an acute inflammatory condition; it probably subsided largely without medicine. If he had been thoroughly treated at that time there

would have been a good chance of his not having this present trouble. Treatment should be with mercury, neosalvarsan, and potassium iodid. Some relief of his bladder difficulty might be gained by a vigorous antiluetic treatment. The prognosis, however, as I have said, is not good.

Case II.—The second case is that of a man thirty-five years of age, a teamster; he entered the hospital on the 13th of March, complaining of a pain in the lower left chest and upper part of the abdomen and a paralysis of the right half of the body, including the face, arm, and leg. His history is as follows:

Six years ago he had sores in the mouth and throat and a sore, leaving a scar, on the left shin. This was after a venereal infection which was not typically characteristic of a chancre.

The paralysis on the right side of his body came on at night while he was asleep, and was associated with pain in the right side of the head. He could not talk at all at first. He has spent the last six years in hospitals—one year at Oak Forest and six months at a hospital on the north side, probably Dunning, then a year or a year and a half at Oak Forest. He spent the four- or five-year interval living with his sister and in the almshouse; has derived most of his support from his sister.

He had pneumonia when twelve years old; does not remember any other illness. He never injured himself badly, except when a child, an ice-wagon ran over his left hand. As he walks, you see he swings his right leg through the arc of a circle, elevating the right side of the pelvis as he does so. The knee is held stiff. The foot is brought down onto the floor with a skuff and a stamp, the heel scraping, and then the body weight is thrown onto the foot rather sharply because of the stiff knee. The right arm is slightly abducted, the elbow slightly flexed, and the palm turned toward the rear. As he turns quickly he loses his balance. He cannot stand on the right leg alone without support. The forehead is wrinkled at command with equal strength on the two sides. The right eye is closed as tightly as the left. In attempting to show his teeth and in strongly protruding the tongue, however, the mouth moves much better on the left side than on the right; the angulus oris is drawn much further to the left and the

left nasolabial fold is much deeper than the right. The tongue, which protrudes in the middle line, shows a fairly coarse tremor. All movements are possible in the right hand and arm, but are executed slowly. His grip in this hand is powerful, but he is slow in exerting it, and he cannot let go, cannot relax quickly. In attempting to make and unmake a fist rapidly there is evident weakness of the extensor muscles, especially of the index-finger.

He had a Neisserian infection several times at intervals of several months, beginning when he was nineteen. His father died at forty. Formerly he drank considerably, but had to quit because his stomach was bothering him.

The plantar reflex is frankly plantar on the left side; on the right side it sometimes is plantar and sometimes extensor, sometimes it holds its position, all of which speaks for involvement of the pyramidal tract. His deep reflexes on the right side are much increased, there is an ankle-clonus and also a knee-clonus on that side. The deep reflexes in the right arm are much increased over those of the left. There is no hemianesthesia. He feels about as acutely on one side as on the other, touch, and pain, and movement in the joints. There is no hemianopsia.

DR. HAMILL (to Patient): What is your name?

(Patient tells full name.)

DR. HAMILL: There is no difficulty whatever about that; he says those two words very well; he is perfectly familiar with them, and there is no disturbance with the muscles of speech.

What are you trying to do now?

PATIENT: Socks.

DR. HAMILL: You see he uses just the one word, "socks," not "putting on my socks."

Can you say "putting on my socks"?

PATIENT: Putting on my socks.

DR. HAMILL: That's fairly well done. Can you say "Peter Piper picked a peck of pickled peppers"?

(Patient repeated this fairly well with prompting.)

DR. HAMILL: Say "Theophilus Thistle the successful thistle sifter."

(Patient unable to repeat this.)

DR. HAMILL: However, in very complex movements, as in "Theophilus," he does not do very well.

Count from one to twenty.

(Patient counts well.)

DR. HAMILL: You see without any disturbance whatsoever he does this.

Now count from twenty down to one—twenty, nineteen, eighteen—go ahead.

(Patient attempts to do this, but fails.)

DR. HAMILL: Can you say the months of the year? Go ahead.

PATIENT: January, February, March, April, May, etc.

DR. HAMILL: Can you say the days of the week?

PATIENT: Monday, Tuesday, Wednesday, Thursday, etc.

DR. HAMILL: Now say them backward.

(Patient tries, but cannot do this.)

DR. HAMILL: I'll say them first (does so); now you say them.

(Patient tries, but shakes his head.)

DR. HAMILL: Saturday, Friday, Thursday—say that.

PATIENT: Saturday, Friday, Thursday (can go no further).

DR. HAMILL: He seems to be perfectly able to pronounce, to speak names, when they are in their natural sequence, but when the order is reversed he is entirely unable to count from twenty down to one, to give months backward, or days of the week backward. He says the Lord's Prayer with great ease, rattles it off without a pause or mistake. When I hold up various common objects before him he names them correctly, some without hesitation, some only after five to twenty seconds. A watch, a chain, a key, a pen are thus named at once. Various parts of his face are named at once when touched. The hands of a watch, a key-ring cause much trouble. When I point to the watch hands and ask him what they are, he doesn't answer; then when I ask him, Have you any? You see he looks down at his own hands almost at once, but still says nothing. Again I ask him, What are these? (pointing to the watch hands), and he says, "fingers," but smiles, colors up, and then says "hands."

We see, then, not a paralysis of the muscles of the arm and leg, and not a paralysis of the muscles of speech, but a failure to

use those muscles easily. He *can* flex his knee, he *can* make a fist, and he *can* say almost any word, but he can't flex his knee in walking. He can't make and unmake a fist quickly and repeatedly, and he can't say one word after another in any but their habitual sequence where they occur in some well-memorized order. There is considerable disturbance in the emotional sphere, as I will attempt to show you.

DR. HAMILL (to Patient): How long have you been sick?

PATIENT: Six years.

DR. HAMILL: Six years—do you feel pretty sorry for yourself sometimes? You have been in an old hospital like this for some time and in an old hospital like Dunning, and sometimes you want to cry and feel sorry for yourself?

(Patient starts to cry.)

DR. HAMILL: Without going any further you see the right side of his face is beginning to come down; tears are in his eyes.

I think we can get you entirely well and you can go out and have just as good a time as you ever did. That would be pretty fine, wouldn't it?

PATIENT: Yes (smiling).

DR. HAMILL: You see it is not at all difficult to cause an expression of sorrow or joy.

This is probably due to an involvement of the putamen of the nucleus lentiformis or of the optic thalamus.

Where are we to place the lesion in this patient and what is its nature?

There is hemiparesis of the right side of the face, the right arm, and leg. This paresis is of a spastic nature, with increased deep reflexes and diminished abdominal and cremasteric reflexes. Because of this spasticity and change in reflexes we must class this as an upper motor neuron disease. Because of involvement of the face we must place it above the nucleus of the facial nerve; above the midbrain.

As the motor fibers leave the motor cortex of the brain, the cortex forming the anterior lip of the fissure of Rolando, they are gathered together in the internal capsule where they occupy a very small space. They form most of the anterior and middle

portions of the internal capsule, while the posterior is largely occupied with the ascending sensory tracts. We have shown there is no sensory disturbance in this man, consequently we may assume if the disease is in the internal capsule it is in the anterior portion. Also the anterior extremity of the internal capsule underlies quite closely that portion of the cerebral cortex controlling the motor mechanism of speech—the posterior end of the third frontal convolution, Broca's convolution. Here the fibers pass between the lenticular nucleus and the optic thalamus. So you see if involvement of these latter basal ganglia is the cause of emotional disturbance, a lesion of the anterior portion of the internal capsule would account for all the symptoms.

As to the nature of the lesion: Its occurrence soon after the appearance of the secondaries of syphilis makes it altogether probable that it was of a syphilitic nature. There is no albuminuria, no high blood-pressure or enlarged left ventricle to suggest its being due to hemorrhage. There is no high degree of atheroma to suggest the late thrombus formation; hence, one can assume it was of the early vascular type, vascular and perivascular, probably a gummatous closure of one of the branches of the middle cerebral artery supplying this region.

If he had had vigorous treatment at the time his trouble first came on he in all probability would have cleared up considerably. But he did not have such treatment, and the syphilitic process has gone on until now there is undoubtedly considerable destruction. There are degenerative processes in the pyramidal tract and these degenerative processes cannot be repaired. Some improvement can undoubtedly be brought about by vigorous treatment, and there is a possibility of stopping further development of other processes.

This treatment will be neosalvarsan, intravenously, at intervals of four or five days; then intramuscular injections of mercury along with large doses of potassium iodid for a course of two or three months. Then another course of neosalvarsan, followed by another course of mercury. Until he has had such a course of treatment it is not fair to say that he has been vigorously treated.

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